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CHAS. R. STORZ

3466 Carrollton Ave., Indianapolis, Ind.

THE LARYNGOSCOPE.

VOL. XXXVIII

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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding
that they are contributed exclusively to THE LARYNGOSCOPE.)

THE ROLE OF SYPHILIS IN EAR, NOSE AND THROAT WORK.

DR. A. T. WANAMAKER, Seattle, Wash.

The incidence of syphilis varies in different countries, different races and according to different statisticians. Lieut.-Colonel Vedder states that in some of the peasant villages of Russia, nearly all the population have the disease. From statements made by several physicians practicing in Alaska, the same is true of the Alaska Indians. All authorities agree that it is more prevalent in the colored race than in the white.

In our practice in Seattle, where we have several thousand Japanese, we have found syphilis more prevalent among them than among the white. Insane hospitals and various state institutions naturally show a higher percentage than army or navy recruits or private hospitals.

The army makes the Wassermann test on all its recruits, and inasmuch as men are taken in from various sections of our country, in all walks of life, it furnishes one with a very fair idea of the prevalence of syphilis in the United States.

In 1915 Lieut.-Col. Vedder published his findings. Of the 1,019 recruits, practically 8 per cent of them gave a 4+ Wassermann. All of these men had a rigid physical examination, and had there been any suspicion of their being syphilitic, they could not have entered the ranks. In addition, there was 9.02 per cent that gave a +3 Wassermann reaction. Therefore, the total number of undiagnosed

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syphilitics that apparently showed no signs of lues would be 16.77 per cent. The Colonel adds, in his private opinion, that the average per cent of men between the ages of 20 and 35 will run at least 20 per cent.

STATISTICS OF LIEUT.-COL. VEDDER.

	Total No. Tests	% Known Cases	% Positive (4 plus)	Undoubtedly Syphilitic	Doubtful (3 plus)	Total % Syphilitic
Recruits	1,019	0	7.75	7.75	9.02	16.77
Cadets	621	0	2.57	2.57	2.89	5.46
Whites Enlisted	1,577	3.44	2.77	8.21	7.87	16.08
Colored Enlisted	1,472	1.08	21.8	22.21	13.11	36.00
Porto Rico Reg.	531	13.55	28.58	42.37	13.55	55.93
Military Convicts	1,145	6.48	9.50	15.98	5.67	21.65
Insane Soldiers	567	3.51	8.29	11.80	7.41	19.21
Tuberc. Soldiers	229	7.56	15.72	23.28	15.72	39.00
Sold. Sent Home	1,171	11.62	13.04	25.02	9.73	34.75

At West Point, where the young men came from the great middle class, all of whom are well educated in order to pass the strict entrance requirements, 3 per cent were positive.

For the years 1925 and 1926, the Wassermann blood test (Kolmer method) has been run routinely at our county hospital. There were a total of 1,739 cases tested; 15.75 per cent were positive.

Also, during the year 1926 the Wassermann test, using the Kolmer method, was run routinely at the Virginia Mason Hospital (a private hospital); 1,150 tested, 3.1 per cent were positive.

Since Aug. 1, 1920, the Wassermann test has been made routinely on every case (except those for minor operations) entering the Swedish Hospital. From Aug. 1, 1920, to 1923, Dr. Nixon, the pathologist, classified the cases as follows: obstetrical, surgical, medical, suspected and unsuspected. The Kolmer technique was the one adopted. Before the inauguration of the routine examination, there was considerable discussion among the staff as to whether it was worth while. Several believed that most cases would be diagnosed clinically. The result speaks for itself. Only 48 out of 171 positive in which the disease was suspected.

STATISTICS FROM THE SWEDISH HOSPITAL.

	No. of Cases	No. Cases Positive	Total %	Cases Not Diagnosed	Per Cent Not Diagnosed
Obstetrical	1,810	8	0.5	9	100
Medical	1,787	72	0.9	60	83
Surgical	3,366	60	1.8	54	90
Questionable		30			
Total	6,963	171	3	123	86

Since 1923 to Jan. 1, 1927, there have been 9,233 cases tested; 3.6 per cent were positive.

This laboratory has done our work for the past ten years, and it is at Dr. Nixon's suggestion that this paper is written.

Right here let me add that one can get more useful information and enthusiasm for his work from occasional discussions with a good laboratory man than from anyone else.

Diagnosis: Inasmuch as syphilis may stimulate practically all of the known diseases, it is possible to discuss only a part of the diagnostic aids, emphasizing some of the commonest, yet often overlooked points.

1. *Pathognomonic Signs:* In a few cases one gets the history of a previous infection, or of a former positive Wassermann test. Others show positive secondaries. That is, lesions, in which the spirocheta-pallida may be shown by dark field illumination.

2. *Suspicious Signs:* In taking the history, one occasionally meets a person who tries to hide, or resents giving you any information about himself, or he may deny some direct question in such a way as to make you suspicious. Little does he realize that his very attitude may make you think that he has the disease.

The history of miscarriages or still births always calls for investigation. On examination we occasionally see the evidence of secondaries, such as:

1: Eruption on face, neck, arms or chest. Either as the characteristic measles copper-colored macules, or the delayed eruption that may assume varied forms.

2: Cracks or indurated areas on the lips, especially those that show no tendency to heal.

3: Syphilitic catarrh—most common during the first six or eight weeks, but which may recur any time during the first two years.

With this, there is usually associated mucus patches, small, white, glistening areas that later ulcerate. They are found any place in the mouth and occasionally in the larynx. The tonsils are swollen, and all mucous surface of nose and throat has a deep red color; although the patient may give no history of an acute cold, the glands in the neck will often be found swollen and tender; hoarseness frequently is present. One should be careful not to remove the tonsils at this time, as there is grave danger of the operator infecting himself.

The patient may complain mostly of being unable to breathe through his nose, in fact there may be scarcely any throat symptoms. *Nasal congestion not relieved by cocain and adrenalin is a point to be noted.* Practically all such cases in our practice have given a positive Wassermann.

4. *Periostitis and Perichondritis:* Symptoms simulating a frontal sinusitis with tenderness over the floor of the sinus and over the brow, extending toward the temple, absence of pus in nose, some mucous membrane congestion, negative X-ray findings, should receive

the Wassermann test. At least you can give them the therapeutic test. Mixed treatment a week, or often four days, will bring relief from all the symptoms.

It has been my practice to put some of the persistent frontal sinus cases on mixed treatment. Pain or tenderness over the bridge of the nose, in absence of history of trauma, is usually of specific origin; likewise the large, swollen, tender nose, usually due to involvement of the lateral or septal cartilages, is mos talways due to lues, occasionally to tuberculosis.

5. *Laryngeal Involvement*: a. I have already referred to the simple catarrh, causing the husky voice, and to the mucus patches that may be on the chords or epiglottis; b. diffuse infiltration; c. gummas; d. ulceration; e. papillary nodular neoplasms; f. perichondritis; g. paralysis; are all conditions that require differential diagnosis.

A point to be remembered with reference to specific infection of the larynx is absence of pain.

6. *Condition of the Teeth*: This is especially diagnostic in the inherited form, but badly decayed teeth with poor condition of the gums, may have syphilis as the underlying cause.

7. *Eye Symptoms*: Interstitial keratitis, according to Stokes, occurs in 52 per cent of patients between the age of 5 to 16 in all cases of inherited lues.

Iritis, or evidence of an old iritis (such as synechia), choroiditis, neuroretinitis, optic atrophy, pupillary changes and muscle pareses are all conditions that may be traced to this disease.

Every man limiting his work to otolaryngology should be able to use an ophthalmoscope, and have sufficient training to recognize fundus changes, and should make it part of his routine examination in all doubtful cases.

8. *Late Secondaries or Tertiaries*: Nodular eruption on the skin of the nose resembling acne rosacea or lupus vulgaris. Two such cases have recently come to our office. Septal ulceration or perforation when located in the bony portion are practically all of specific origin; when in the cartilage, a certain portion are due to it.

Ulcers and gummata may occur anywhere in the nose, throat or tongue. Old scars and adhesions of the soft palate are usually traceable to such. The use of a probe in examining the interior of the nose is very helpful in locating soft bleeding areas, or detecting loose or roughened bone.

9: Loss or distorted sense of smell is often due to lues.

10. *The Internal Ear*: The eighth nerve is the commonest of the cranial nerves to be affected. It may be involved early or late in the

disease; both branches occasionally, but much more often the cochlear alone.

Before the patient complains of any real loss of hearing, tuning fork tests will show a loss for the high notes, or better yet, the audiometer will show a significant curve.

According to Isaac Jones in his textbook on Vertigo and Dizziness, much can be learned from testing the labyrinth. There is no doubt that this procedure is helpful if the tests are properly made, and one is able to properly interpret the result.

Syphilitic Syndrome: In this division I place those cases that show no positive neurological findings, but complain of inability to sleep or headache at night, transient dizziness, tiredness on the least physical or mental exertion, numbness in the hands or feet, drawing sensation in the muscles, or tender sore spots on the scalp, are some of the commonest complaints that may properly be due to syphilis. Generally you will find some of the special sense organs involved, such as sight, hearing, disturbed sense of touch, balance, some of the visceral crises, and altered or lost reflexes.

Serological: It is estimated that between 60 to 70 per cent of cases give positive blood Wassermann reaction. In the hereditary form, nearly 100 per cent. Some observers believe that there are certain phases or stages of the disease in which the blood test will be negative. In syphilis of the nervous system, the blood may not show any reaction but the spinal fluid gives a positive test.

Ventricular Fluid: This may also be positive when the spinal fluid is negative. Or, it may be found negative, and the spinal fluid positive.

Nonne speaks of four reactions in connection with syphilis of the nervous system: 1. Positive Wassermann of the blood; 2. increased globulin; 3. lymphocytoses; 4. positive Wassermann of the cerebrospinal fluid.

The cerebrospinal fluid, when the nervous system is involved, shows: 1. Increased pressure; 2. an increase in the contents of the protein material, and the number of the cells, principally leucocytes (Levinson).

Langé Colloidal Gold Test: All forms of syphilis of the nervous system gives positive reaction in syphilitic zones, the reaction varying with the type of the disease.

Spinal Puncture: There is no question but what this procedure is not done often enough. How often do we see middle aged men with considerable deafness who have passed through various clinics and yet have been unable to find the cause?

When the importance of the diagnosis is emphasized, and the consequences discussed frankly, I believe there would be little difficulty in obtaining consent for the test. Such cases should receive the sodium or potassium iodid, instead of arsphenamin. It is not wise to take the spinal fluid without the patient knowing exactly what is being done, as one lays himself liable to lawsuit. Nonne relates his experiences and advises against it.

Provocative Tests: Kolmer and others advocate the giving of two doses arsphenamin or of sodium iodid intervenously, before making the test. Cases showing involvement of the eighth nerve may get what is known as Herxheimer reaction or shock, due to the arsphenamin, which may cause total deafness.

Can an injection of arsphenamin provoke a positive Wassermann in the nonsyphilitic?

We will quote from an abstract of the paper of H. Boas, and A. Kronmeyer (Ungesk f. laeger, 3-29-23).

"They gave injections of old arsphenamin to seven; neoarsphenamin to six; silver arsphenamin to 26; neosilver arsphenamin to 11. Patients were all nonsyphilitic. Each case was given one to five injections and the Wassermann test carried out once a week. It was invariably negative, whether carried out before, during, or after treatment."

Treatment: When once the diagnosis is made, I believe the patients should be referred to a capable internist, dermatologist, or genito-urinary specialist for treatment, having the patients report occasionally to us to note progress.

No surgical operation should be undertaken on untreated cases of syphilis, unless in emergency.

CONCLUSIONS.

1. There are many cases of syphilis not diagnosed.
2. Routine Wassermann tests in our hospitals is a good measure.
3. It should not be limited to major operative cases.
4. Spinal puncture should be performed more frequently.
5. The therapeutic test should be used to a greater extent.
6. The ear, nose and throat specialist is in a strategic position to diagnose syphilis.

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OTO-RHINOLOGICAL HEMORRHAGE AND SOME METHODS OF CONTROL.*

DR. PHILIP S. STOUT, Philadelphia.

Human blood is a liquid, red in the arteries, blue in the veins, of varying composition, surging through a closed system of vessels at a rapid flow, making a complete circuit in about 20 seconds. It gives nourishment to all parts of the body and carries away the waste matter and is absolutely necessary to life; and not only must it flow but it must be under a certain amount of pressure or life will cease to exist, as for instance, a person dying from profound shock without loss of blood from the vessels.

Blood is made up of two principal parts; the fluid or plasma, $6\frac{3}{4}$ parts; solids or cellular element, $3\frac{3}{4}$ parts by weight. The amount of blood varies with the age. In an infant 5 per cent of the body weight is blood, in the adult about twice that percentage of body weight is blood.

Not all of this blood is in the blood vessels but is distributed as follows (Brubaker's Physiology, 1925 edition): Heart, lungs, arteries and veins contain about one-fourth; liver contains about one-fourth; muscles contain about one-fourth; other organs contain about one-fourth. So that a child having, say, a quart of blood (1,000 c.c.), only one-half pint (250 c.c.) is in the vessels, and an adult having six quarts of blood (6,000 c.c.), only three pints (1,500 c.c.) is in the vessels.

Of course in case of hemorrhage the blood in other parts is rapidly drawn upon and supplied to the diminishing stream in the vessels and yet it must take time to get a very large amount. A horse weighing 1,200 pounds will give 60 pounds, or 5 per cent, of body weight when he is bled to the last drop at one time and this is, of course, absolutely fatal. Only about 1 per cent can be taken with safety. Then there are other factors; for instance, a slow trickle or persistent ooze seems to be more dangerous to life than a large, frank hemorrhage.

Blood cells are roughly red blood cells, white blood cells and platelets. These cellular elements contain: water 200 parts, solids 128 parts. Solids are: hemoglobin 116 parts, other organic matter 10 parts, salts 2 parts. Blood plasma contains: water 604 parts, solids 68 parts. Solids are: fibrin 7 parts, albumin 52 parts, fat 1 part,

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other organic matter 3 parts, potassium and sodium salts 4 parts, calcium and magnesium 1 part.

Normal blood is usually slightly alkaline in reaction. It is designated as the Ph of the blood as compared with water, which has logarithmic notation of Ph 7.35; that is, water is neutral expressed in H+ ions (acid) to 1/10,000,000 of a gram of H+ ions per liter. Ph 6 represents the acidity of 1/1,000,000 and Ph 8 represents the alkalinity of 1/1,000,000. The reaction of blood lies somewhere between the neutral solution (water) and Ph 8.

Blood Clotting (Coagulation) and Bleeding Time. For our knowledge of blood clotting much credit is due to: Woolridge, 1893; Morawitz, 1903; Leo Loeb, 1909; Borden, 1912; Howell, 1916; Mills, 1926; and others. The clotting of the blood: the power of the vessels to retract into the tissue and the lowering of the blood pressure are the factors that make surgery possible, otherwise nearly all patients operated upon would bleed to death.

How is a blood clot produced? Much blood has flowed from vessels since the first formulas were given as to the process of blood clot formation. From 1904 to 1908 I pottered around in one of the large pathological laboratories of this city (Philadelphia) and during that time I occasionally assisted Dr. Leo Loeb in some of his experiments, among which was the use of a substance from leeches, Hirudin secreted in the mouth of the leech, to prevent blood from clotting. As you know, a leech will suck blood until its body walls are stretched to their absolute capacity, which is about three or four times its normal size, and when it is full it drops off and is helpless, dormant, and apparently at peace with all the world. But drop it into a salt solution and it will become very active and will vomit almost every bit of blood it has ingested. The blood is in a liquid state, without any clots, because the leech has in its body this anti-clotting substance which will keep all the various bloods that I saw used for the experiment in a liquid state for a long time. I believed then and do now that such an anticlotting substance is everywhere in the human body where the blood is intended to go and remain in a liquid state. This may be the substance now known as antithrombin.

Some of the constituents known to be in the blood plasma and cells are prothrombin, cephalin, salts, magnesium and calcium, fibrinogen, globulin, albumin and heparin. Prothrombin is acted upon by cephalin from blood platelets and white blood cells to produce thrombin; then in some way with the addition of calcium salts combined with fibrinogen, fibrin is produced; hence the clot. Not as simple as it would seem at first sight, but indeed rather complex, hence our difficulties. Clotting at the point of exit of the blood from the vessel

will be interfered with if there is too much antithrombin or not enough prothrombin, cephalin, calcium or fibrinogen. Each has its role to play to suddenly change the blood from a liquid to a semi-solid, strong enough to overcome the pressure of the blood back of it; in short, to cork the vessel. Other conditions which interfere are high blood pressure, atheroma, arteriosclerosis, jaundice, menstruation, anemia, leukamia, syphilis, severe malnutrition and any debilitating illness or fibrous scar tissue, and in our work a piece of tonsil or adenoid or a large clot left in situ.

Clotting of blood should occur from $4\frac{1}{2}$ to 9 minutes with the usual methods of testing, above this it should be viewed with suspicion and further examination made of the patient before the operation is performed. Heparin is supposed to influence the antithrombin formation.

Bleeding Time. This is still on the border land of the unknown. A patient's clotting time may be as low as $3\frac{1}{2}$ minutes and yet the bleeding time may be indefinitely prolonged. Usually, however, low clotting time is associated with a corresponding low bleeding time, providing, and this is especially true of tonsillectomy, that no sharp, razorlike, cutting edge is brought in contact with the tissues, outside of the capsule of the tonsil let the separating be done with a dull dissector or a not too fine wire snare, gradually and slowly drawn together so that the vessels are torn rather than cut, and under these circumstances the likelihood of a primary or secondary hemorrhage is much less than if a sharp blade is used, whether for loosening the tonsil from its bed or severing the pedicle.

As a simple illustration, all of us have cut ourselves with some old dull penknife. In a moment or two the bleeding was over. On the other hand the new razor blade accidently makes a minute cut and it will bleed and bleed until you lose your temper and the time you have set apart for your breakfast. The cut the old knife blade made was a laceration with the incision, the new razor blade made a clean incision, with not much if any laceration, hence the influences, such as the torn ends of the vessels and the juices from the contused tissues in the immediate neighborhood were lacking, and therefore the clotting is delayed at the point of exit, although the blood may clot promptly as it drops into a vessel. That is, the clotting time of the blood may be normal but the operative procedure has changed the bleeding time. This is also true of adenoid removal when using an instrument of the Guttstein type, so that one might say that, other things being equal, the sharper the instruments are which come in contact with the tissues outside of the tonsils and adenoids the more likely you are to have operative and possibly postoperative hemorrhage. Other factors that have to do with bleeding time are leaving

of pieces of tonsil and pieces of adenoid tissue; the cutting of a vessel lengthwise so that it cannot contract; the injury of a plexus of veins at the base of the tonsil; more rarely, an injury to the posterior end of the nasal septum or turbinates; or the cutting of an anomalous branch of the sphenopalatine artery; or a large ascending pharyngeal artery coming off from the bifurcation of the common carotid into the external and internal carotic; injury to the internal carotid artery; the formation of a clot in the postnasal space or in the tonsillar fossae; persistent hawking of the patient; excitability, anxiety and restless tossing; occasionally goiter; any constriction about the neck; and, no doubt, there are other factors that affect the bleeding time.

Case: A woman, age 40 years, had a goiter for some years. She had peritonsillar abscess, as she said, every six weeks. I know that she had it four or five times a year for years. Finally I consented to operate, tonsillectomy, but with much misgiving. She bled little at the time of operation but oozed later. Twelve hours later she died, apparently from shock induced by bleeding and from the goiter (hyperthyroidism). The hospital was poorly equipped for such a case.

Frequency of Hemorrhage in Ear, Nose and Throat Patients: Ear operations rarely give any serious trouble with hemorrhage. In mastoid operations, if the sigmoid sinus is opened, the hemorrhage is fairly free but I have not found any great difficulty to control it at the time of operation or postoperative. There have been a few cases reported of fatal hemorrhage following incision of the drumhead; however, this is quite rare. It is supposed to be due to incising the jugular bulb. The use of iodoform packing will control hemorrhage in the region of the ear. Hemorrhage occurs at times with fracture of the base of the skull. It is not likely to be very serious as far as the bleeding is concerned.

Nasal Bleeding—Epistaxis: Nonoperative, including malignancy and other neoplasms. Operative and postoperative nasal bleeding is quite common. Causes: Traumatic blow or blowing of nose too violently; hypertension; ulcer of septum (tuberculosis, syphilis and other causes); fingernail cut; operative or postoperative; heart disease; malignancy and occasionally benign neoplasms; typhoid fever and other infectious and eruptive diseases; kidney disease; anemia; hemophilia; scurvy.

Treatment of Nasal Hemorrhage: Semirecumbent posture. If slight, apply cold to forehead and back of neck. Get rid of all clots from the nose by blowing the nose or syringing with hot water. Absolute quiet. If bleeding continues apply some styptic to bleeding point, providing you can see it; if unable to see the bleeding point, pack with gauze or cotton, using either peroxid, epinephrin or eph-

drin, a weak solution of cocain—this is best combined with epinephrin solution. If it continues, put in a posterior pack, using a soft rubber catheter to draw it into place. I would suggest that some of the drugs and preparations mentioned in this paper be given in conjunction with the local nasal treatment. One other thing I desire to emphasize and that is, in bleeding from the posterior nares, gargling and flushing the posterior nares by way of the mouth with right hot water has saved several of my patients.

Case: An athlete, a case I have already reported. Following a hand stand spring began to bleed from what I now believe was the sphenopalatine artery. He had been bleeding more or less persistently for two days and two nights. Nothing seemed to help this condition. Then we tried right hot water and the bleeding ceased. Of course, the next thing we would have done would have been the ligation of the external carotid artery, which must be borne in mind in very extreme cases. Death may occur from nasal bleeding. I saw one such case while I was an interne.

Case: A colored woman, age 45 years, came into the hospital with a history of more or less persistent nose bleed for several days. She was put to bed and we used the usual methods and remedies to control hemorrhage that we knew at that time—21 years ago—but she continued to bleed no matter what we did; the various things that had been suggested by the chiefs who had seen her and the things we found in the textbooks, but the morning came and she passed away. It was indeed a shock to us all. A patient to die from simply a nose bleed. This fatal case early in my career was of the greatest value to me, although during the years since I have seen many cases of hemorrhage, great and small, I have ever been on the alert and never treated them with indifference or unconcern. The possibility of death seemed always imminent.

Tonsil and adenoid hemorrhage is usually either operative, post-operative, or malignancy and the man who says that he has done a large number of tonsil and adenoid operations and has not had any severe hemorrhage either at the time of operation or postoperative is a fit candidate for the Ananias Club. In adult and child clinics combined, the number of severe hemorrhages in this operation is about 4 to 6 per cent.

Symptoms of Hemorrhage: Presence of blood in nostrils or throat, mucus and saliva from mouth blood-streaked, throat looks moist, at times there is a frank bleeding from a vessel which can be easily seen, a clot may be on the spot from which the blood is oozing, bluish-red line along the edge of the soft palate and pillar if oozing beneath clot. Subconscious person is continually swallowing. Nausea and vomiting of either dark or at times dark red blood, mixed. This

may be the first warning of a bleeding patient. Pallor, pale lips, whites of eyes more prominent. Lowering of blood pressure. Increase of pulse rate, may be weak, very rapid, finally imperceptible. Irregular breathing (Cheyne-Stokes). Temperature in adult may be subnormal, in child it rises with the loss of blood up to 105° rectal just before exodus. Ringing in the ears, sound of bells, head noises. Sighing, yawning. Air hunger. Pain in region of stomach. Restlessness, talkative. Anxiety.

Laboratory findings: Increase in leucocytes, especially in polymorphonuclear and myelocytes. Decrease in hemoglobin.

Recently Wilson and Johnson, working at the University of Pennsylvania, have shown that there is first an increase in the alkalinity of the blood and then a fall below normal.

Treatment of Hemorrhages: Treatment should begin as soon as you first see the patient, even if it is weeks or months before the operation you should ascertain whether the patient has any history of having any trouble to stop bleeding after tooth extraction, following cutting of finger, lancing of quinsy, or a history of bleeders in the family. This, I am sure, has saved me from several fatalities. So also a simple clotting time can be easily made in the office or simply put the patient on calcium medication. It certainly seems to be of value. Just before the operation have the clotting time made. There are several preparations that can be given orally: fibrinogen, ceanothyn, hemostatic serum. Whether it is right to risk a hypodermic of some animal product as a routine I am not prepared to say; the risk seems to me to be greater than the necessity. An adult's blood pressure should be taken, and possibly a Wassermann.

Don't deplete or starve your patient too much before operation. Urine examination always for general anesthesia patients. During the operation use dull instruments whenever possible, especially in tonsil and adenoid work. Have the patient thoroughly anesthetized, whether local or general. In local work do not use too much epinephrin; it will often be followed some hours later by hemorrhage.

Have the patient leave the table with a dry field. To accomplish this: pressure with soft gauze or cotton sponges. Locals use hot water gargle, applications of saturated solution of tannic and gallic acids in alcohol carefully applied so that it does not get down into the larynx, pick up the bleeding point with a hemostat and, finally, stitch tie, if necessary.

The placing of the stitch tie in the tonsillar fossae is not as easy as it seems to be, especially at the lower angle. I would suggest that the operator stand in the position of the anesthetizer and use long, straight needle holder with a quarter-curved, small, fairly stout gold needle, using No. 1 catgut. Continue with the stitches until the field

is dry. Occasionally this is not possible. Under those circumstances it is advisable to give an anesthetized patient some of the preparations used hypodermically and treat the bleeding fossae by placing iodoform gauze in the fossae and put two or three stitches over the gauze from one pillar to the other. In my experience this has usually checked the wretched bleeding and oozing that fortunately only rarely occurs. Leave the iodoform gauze in for 24 hours, then remove carefully. If the bleeding recurs, replace it once more. I have only had to replace it once. That was when I removed it in eight hours.

Some Methods that Have Been Used to Control Postoperative Hemorrhages: Inject salt solution into the tissue of bleeding fossae. Inject local anesthesia solution into tissue of bleeding fossae. Inject 2 or 3 minims of adrenalin chlorid solution into the fossae, which will produce an ischemia. This will bring into bold relief the bleeding point, which can be dealt with as the operator sees fit. Put a number of hemostats on the bleeding area and allow them to remain for some time. Local applications, oral administrations, hypodermic injections and intravenous injections of various substances will be mentioned under drugs and animal products. Enteroclysis. Transfusion. Pressure with Clendenin or Mikulicz's clamp. Pressure with thumbs or fingers on the bleeding area with a pad of gauze.

Case: W. L., male, age 20 years, 1910, had one tonsil removed with tonsillotome. Began to bleed two hours later. When I was called 1½ hours after the bleeding had begun I found him almost bloodless and pulseless. Nothing that we could do seemed to check the bleeding. Finally I put my thumb with a piece of gauze and pressed it against the tonsil fossae. It stopped bleeding. Later he was sent to the hospital, where five hours later he again began to bleed. After one hour of using other methods they also resorted to this thumb pressure and controlled the bleeding in about one hour. He had no other recurrence. I did not remove the other tonsil.

Permit me to say a word about responding to the call for help from a brother practitioner, assistant, or interne. Words fail me when I try to express my contempt for the one who refuses to come to the aid of another who is sweating blood trying to save a patient from bleeding to death, or death from any other cause for that matter. Dr. Chevalier Jackson, spending his nights on a cot in the ward, watching with the interne the weak breaths of his little patients, is the answer to anyone who feels that he is so high and mighty that he must not be disturbed even if death is hovering over the patient.

Management of a Patient: See that the patient leaves the operating room with a dry field. Have him lie on the right side, left leg drawn up, face down. This will permit any blood or saliva to flow from the mouth or nose. Ice collar to neck, except very young children.

Watch temperature, pulse and respiration. Nurses should receive more training along this line. If the patient is recovering from ether, notice if he is swallowing frequently. Notice if there is any undue pallor, bloodless fingernails, color of lips, whites of eyes, condition of pupils, sighing, air hunger. Notice the amount of vomitus and frequency. Undue thirst. Restlessless. Chilliness. Complaints of pain in the stomach. Talkative, whining. Nervousness. If the patient has lost much blood, see that his head is not raised above the level of his body, it may cause instant death. These things should be kept constantly in mind by those having charge of the patients.

In short, a great preventative for fatal hemorrhage, or, for that matter, the fatal outcome for any other reason, is a well appointed hospital, equipped with the modern appliances necessary to detect and control serious conditions during and following operative procedures. In fact, preoperative, operative and postoperative care can be more thoroughly carried to a successful conclusion in a hospital under such ideal circumstances. Some of the recently built hospitals have been constructed somewhat along these lines.

There is still an ideal which may eventually come to this city, and that is a hospital devoted entirely to eye, ear, nose and throat; oral and plastic surgery; head and neck surgery, especially the surgery of the thyroid; bronchoscopy and esophagoscopy; the study of allergy, manifested as hay fever and bronchial asthma.

The wonder is that such a hospital has not been established in this city years ago. In such a hospital each would be a kind of specialist; the nurses, the internes, the assistants to the chiefs, and the chiefs; with nothing to detract them from the study and care of these special cases. Also, with the large number of patients, a wonderful opportunity would be afforded for the graduate student and others who might desire to follow some investigation. The possibilities of such a hospital are limitless. Incidentally, I would suggest that a library and proper laboratories for these special studies be combined with the hospital and open to all who are ethical physicians.

Case of Poorly Conducted Hospital: A child, age 6 years, was taken by its mother to a hospital which at that time used any available room to operate for tonsils, a surgical dressing room or side room of a ward. This child had its tonsils and adenoids removed and following the operation it developed diphtheria of both eyes and died. I happened to know of this case since the child attended a nursery which was under my care. Also, it may be interesting to note that in one of the large hospitals there was a rule within the last five years that the visiting laryngologist should treat the larynx, and he did not have the privilege of the operating rooms, and when an operation, such as a tracheotomy, was indicated, the laryngologist

had to get permission from the patient, his near relatives, the medical or surgical chief, and finally the superintendent of the hospital. Usually before all this was accomplished the patient passed out.

Various Drugs and Animal Products Used in Control of Hemorrhage. Local Applications: Hot water gargle. Gallic and tannic solution, alcoholic. Solution of silver nitrate. Epinephrin solution (adrenalin solution). Ephedrine. Cocain solution. Peroxid of hydrogen. Alum. Aluminum acetate. Iron preparations. Chromic acid bead. Actual cautery. Iodoform gauze. Ice, externally. For nose bleeding a very old but very good remedy is salt bacon or ham fat put in the nostrils. It sometimes acts like magic.

Oral Administration: Large dose of calcium. Ceanothyn. Fibrinogen. Hemostatic serum.

Hypodermically (not intravenous preparations): Salt solution. Digitalis for hypodermic use. Fibrinogen. Epinephrine (adrenalin chlorid solution). Coagulose. Coagulen. Para-Thor-Mone (parathyroid extract). Kephalin or thrombinplastin.

Enteroclysis: Salt solution to which glucose may be added.

Sedative: Codeine. Morphin and atrophin. Bromids. Some of the more recent coal tar products.

Intravenously: Salt solution, .7 to .9 per cent (neutralized gum acacia is sometimes added). Human blood, transfusion, properly typed, 250 c.c. to 1,000 c.c. Horse serum (beware of patients suffering from asthma). Calcium chlorid solution. (When in doubt do not inject anything into the veins that may cause clotting of the blood in the vessels. It may prove fatal.)

Supporting Drugs: Hypodermic injection of some iron and arsenic preparation.

Have Emergency Grip Always Ready: I would suggest for your own peace of mind that you always have ready a grip which will contain the things you may need to control a sudden postoperative hemorrhage for which you may be called upon to treat. While it is best to get the patient to a hospital as soon as possible, if the bleeding is severe, yet it is necessary at times for you to act very promptly or your patient may not reach the hospital alive. Also, I have found that at times the hospitals were illy prepared to handle such an emergency and I have used my own instruments and medicaments. I have never regretted having them all in one bag and with me. Two or three o'clock in the morning is a bad time to obtain anything from the pharmacy. The contents of this grip I will leave to your individual judgments; however, have an outfit for tracheotomy, acute edema of the larynx may make this operation an immediate necessity. Be prepared.

Medical Arts Building.

ONE OF THE OPERATIVE TREATMENTS OF OZENA. PRELIMINARY COMMUNICATION.*

DR. K. B. STEINMANN, Leningrad, U. S. S. R.

Rational therapy is grounded on the etiology of disease; however this thesis cannot be applied to the treatment of ozena, the etiology of which is as yet quite unknown.

In the course of the last years surgical methods have risen to the surface of the therapeutic tide of ozena, and through want of something better, we are obliged to confine ourselves to them.

Many means have been suggested lately for the treatment of ozena, but none of these has brought about any good results. In some cases there may have ensued a certain improvement, but it does not yet signify recovery, and on the other hand, how great are the sacrifices it requires! In most cases we see only a fleeting effect, and the ozena, that seemed to wither for a certain time, unfortunately much too short, blossoms once more in stinking flowers, with all its dryness, its crusts, its painful feelings, and patients seek again for help, willing to bear any tortures and trials in order to get rid of the heavy disease that pursues them at all times and places.

I suppose, that when choosing surgical methods for treatment, we should take into account the two following considerations: 1. the effect of the obtained result in the sense of the duration of the action, as well as the amount of success; and 2. the sum of the patient's feelings, *i.e.*, the seriousness and weight of the operative intervention.

In fact, the question should be viewed exclusively in this light and this criterium ought to be applied for the choice of the one or the other operative method.

Just in the same sense as all ways lead to Rome, all the operative methods for the treatment of the ozena known until now tend to the same purpose; however, all operations are not equally accessible to the wide circle of specialists; and operative methods differ from one another by the seriousness, as well as by the length of their performance. Thus, Lautenschläger's operation, very cumbersome and dangerous in itself, has not been generally approved of, but has acted as stimulus to the search of new operations, more simple as to performance and less tedious as to time. Halle's modification, technically

*Report made at the meeting of the Leningrad Otolaryngological Society, Oct. 14, 1926.

*This patient, operated in 1925, has left Leningrad, having thus rendered observation impossible.

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more simple and requiring less time, has proved much more convenient, is more easily performed, and is, therefore, preferred by surgeons. Brünning's and Schönstadt's operations, being rather unsuccessful modifications of Lautenschläger's fundamental one, are less secure and not so easily performed as Halle's, and have therefore less adherents amid specialists, so much so that to my knowledge they are now utterly rejected. The same fate has overtaken the operation proposed by Prof. Wittmack even in case of a complementary fistula in the alveolar apophysis of the upper maxilla.

Dr. Bourak has proposed a less cumbersome and more convenient operation in the sense of therapeutics, "My Method for Surgical Treatment of the Ozena", *Zeitschrift für Ohrenheilkunde*, 1924, which deserves the attention of otosurgeons. The operation is performed thus: an incisure is made under the upper lip, reaching the aperture; the mucous membrane at the bottom and on the lateral side of the nose is separated underneath and above the interior nose-shell, *i.e.*, that it is cut into a hollow with a scoop until it reaches the choanae. The mucous membrane with the nose-shell is pushed aside to the septum, thus forming a pocket into which vaseline oil is poured; after this the wound is sewed up.

The simplest method as to technic is that of Eckert Moebius, *Zeit. für Ohrenheilkunde*, Bd. VII, H. 1, who inserts between the mucous membrane and the cartilage of the septum a boneplate, carved out of the knee pan of an ox (the plate is prepared in the following way: the bone is steamed out in water, then kept for some time in 10 per cent potassium, after which it is dissected into discs of the desired proportions, and these last are put into the nose). The wound is not sewed up. The author has operated in this manner 20 patients, of which 17 with very good results; in three cases the bone, which was beginning to rot, was falling out, though without bad consequences for the patient. Length of observation from 2 to 20 months.

It is clear that an intervention of this kind is the easiest and that patients bear it better than any other. The results are not bad compared to other methods, but this way is much simpler than any other. Its defect lies in the preliminary fashioning of the bone, its possible unsteadiness and its resorption in a shorter or longer lapse of time.

Not being acquainted with the operative method of Moebius, I made in 1925 in a case of an undistinct ozena the following operation to a woman age 24 years. I inserted between the mucous membrane and the cartilage of the septum a plate of cartilage taken from the nose of a man operated just before because of a deviation of the septum. The technical details of the operation are the following:

the usual incision of the mucous membrane of the septum, such as is applied in the typical operation of Killian; careful separation of the mucous membrane with the perichondrium down to the glottic end of the septum; in the pocket thus formed, insertion of one or more plates of a human bone cartilage, absolutely fresh, taken from the septum of the nose of a patient, recently operated because of its deviation. The wound is sewed up once or twice (the last is not compulsive). During the operation the strictest aseptic must be observed; when dealing with the cartilage the latter must be sustained with an instrument (not with the hands); before being placed into the pocket of the septum the cartilage must be kept for two or three minutes in a solution of rivanol, 1:500, then bathed in saline solution, carefully introduced into the pocket of the septum and pushed on to the choanae. The first plate is followed by a second; in this way the septum is made to approach the lateral side of the nose to the distance of the breadth of the cartilage. If the cartilage is very thin, it may be bent in two or two plates may be joined, putting one upon another; but the greatest attention must be paid to the atrophied mucous membrane of the septum, as its rupture as well as its careless separation at the place of the incision may bring about the entire annihilation of the work.

My collaborators and I have operated in this way 14 patients, 10 women and 4 men, from 17 to 48 years, with the following results: very good in 4 cases (28.6 per cent, women), satisfactory in 5 cases (35 per cent), and no results in 5.

In 6 cases (42.8 per cent) sutures have been made, which renders the operation longer, but accelerates the cicatrization of the wound. In one case there ensued a suppuration, abscess on the septum, 1½ months after the operation; this required the removal of the septum. This patient suffered at the same time of erysipelas on the face, which had a happy ending.

I advised to insert the cartilage only from one side and not to set about the second until the first has grown steady.

Maximum of observation time, six weeks,* if during this time the results keep sound.

Between the operated patients there were three who had undergone an operation after Halle's method one or two years previously to the present one; the results had been unsatisfactory in all three cases and the patients returned once more to the conservative treatment.

The convenience and advantages of the surgical method I propose are evident; the facility of its performance is beyond doubt; the results are just as fortuitous as it is impossible to foretell them in any other operation of the ozena.

MYXO-PAPILLOMA OF THE NASAL WALL IN THE SPHENO-ETHMOIDAL REGION; CASE REPORT.*

DR. A. LOBELL, New York City.

This case is hereby reported for the following reasons: 1. The growth assumed unusual proportions and location. 2. An attempt to remove it completely proved unsuccessful in other hands. 3. The results obtained by its radical removal, followed by radium therapy, seems to have checked its recurrence satisfactorily.

E. G., No. 431,679, was admitted to the Manhattan Eye, Ear and Throat Hospital on March 10, 1926, on the service of Dr. John E. MacKenty, under the care of the writer. His family and previous history were irrelevant.

Present History: He was a Russian Hebrew, age 57 years, and a tailor by trade. In the latter part of 1923, he noticed a gradual onset of left nasal obstruction. In May, 1925, an operation was performed on his nose. He states that pieces of tissue were removed from his left side. After that, his nasal obstruction lessened in degree, but the improvement was only of short duration. Within six months prior to admission, the obstruction has gradually increased to complete occlusion. He suffered continuously from a dull headache, which radiated over the left fronto-parieto-occipital regions. His olfactory function was considerably impaired on the left side. He had a moderate amount of an odorless mucopurulent discharge of both nasal fossa. There was no history of epistaxis, ocular disturbance or loss of weight.

Examination: He looked anemic, moderately debilitated and much older than his real age. Anterior rhinoscopy revealed a pale mucosa in both nasal fossa. The left middle turbinate was not identifiable. The middle meatal region was occupied by a uniform growth. It was immovable and hard to the touch of the probe. Posterior rhinoscopy showed that the left choanal orifice was occluded by a mass of tissue. Finger palpation proved it to be a hard mass and there was no bleeding after manipulation.

Laboratory Reports: The blood Wassermann was negative. The X-ray of the sinuses showed as follows: Right frontal, moderate involvement. Left frontal, large and deep, moderate involvement

*Read before the Lebanon Hospital Alumni Society, November, 1926.

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from granulations; both ethmoids, moderate involvement, with some absorption of the cell walls; left antrum, moderate involvement; right antrum is clear; sphenoids are very large and clear.

Operation: Under local anesthesia a submucous resection was performed in order to gain working space. After removing a great deal of hyperplastic tissue around the middle turbinate and resection of the latter, a large tumor mass was peeled away in toto from the walls of the sphenoethmoidal regions (Fig. 1). This tumor was firmly adherent to the walls of the sinuses. Deep indentations of the walls were seen in the bed where the tumor was lying. Exenteration of the ethmoidal cells and partial removal of the anterior wall of the sphenoid showed that the tumor did not invade these cavities.

Macroscopic Appearance: It was multilobular, resembling omental tissue, and some of the convolutions looked not unlike those of a cerebrum. It was of hard consistency. It was pale, with a yellowish tint in some areas. It measured approximately $4 \times 2 \times \frac{1}{2}$ -in.

Microscopic Report: Dr. A. A. Eggston reported as follows: The specimen consisted of several cauliflower-like masses. Sections show a marked hyperplasia of a stratified cuboidal epithelium resting on an edematous fibrous bed, with areas of inflammatory infiltration. The diagnosis is myxopapilloma. This type of tumor has a tendency to recur, but usually it does not metastasize.

Course: The convalescence was rather stormy, owing to his general weakness and debility. About four weeks after the operation he began to improve. A rhinoscopic examination at this period revealed an elevated tissue growth in the ethmoidal region, which looked entirely different from the granulations in the vicinity. In order to give the patient the benefit of the doubt, he was referred to the radium department of Dr. Allan Robinson for his opinion. He decided to give him several applications of radium within a period of four months. After that, the examination showed that the growth had disappeared. The breathing was normal. The general health improved considerably.

Up to the present time there is no apparent recurrence. He will be kept under further observation.

1420 Grand Concourse.

VELOCITY SEDIMENTATION TEST (FAHREAU) IN OTOTOLOGY.*

DR. ARTHUR WEISS, New York.

The accelerated sedimentation of the red blood corpuscles in the bloods of patients sick with infectious diseases has been known for many centuries. Amongst the ancient Greeks it was known as the *crusta inflammatoria* or buffy coat. Galen is supposed to have referred to it as the *crusta phlogista*. It was Hunter, however, who in 1797, first delved into this phenomenon scientifically. He noticed that the red blood cells dropped very rapidly in all acute infections. He noticed further that this sedimentation was fairly proportionate to the amount or severity of the infection, thus if the infection were slight the sedimentation was slight; if severe, marked. Having thus connected the amount of sedimentation with the severity of infection, he sought to determine which of the components of the blood, the red blood cells, or the plasma, carried the causative agent for this phenomenon. He, therefore, mixed the erythrocytes of normal individuals with the plasma of patients suffering with inflammatory disease and found that the red cells sedimented just as rapidly as the patient's own cells. When he mixed the cells of a sick patient with the plasma of a normal person the cells would not sediment rapidly. Thus he placed the causative factor for increased sedimentation in the blood plasma. Before his time, the general impression had been that its cause was something present in the cells.

The work of Hunter was continued by J. Muller, Davy and others. At about the middle of the nineteenth century we again hear of it. It became the topic of prime importance. It served as the source of many lectures and scientific articles. We, then, hear nothing about blood sedimentation until 1917, when Robin Fahreaus started investigating this problem in pregnancy. It had been forgotten so thoroughly that he thought he was discovering something new. He had no idea that he was only reviving an observation known to Hippocrates, and that he was only producing an improved and scientific method for measuring this phenomenon.

Technique: The appearance of this improved means for determining the amount of sedimentation was immediately followed by enthusiastic research throughout the medical world. This soon resulted

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in the advocacy of other methods and modifications. These can be divided into: 1. distance; 2. time; and 3. graphic methods. In all of these methods the mode of procuring the necessary blood is more or less alike. A sodium citrate solution, $2\frac{1}{2}$ to 5 per cent, is used in the proportion of one part citrate to four parts blood. Thus with .4 c.c. of citrate prepared in a 2 c.c. syringe, blood is drawn by venupuncture up to the 2 c.c. mark. It is in the registering of the results, and in the types of tubes employed that the differences demonstrate themselves. In the distance methods (Fahreaus, Westergren, Fischl, etc.) the blood is transferred into a graduated pipette or tube with an inner bore of $2\frac{1}{2}$ -4 m.m. and 20-30 c.m. long. After $\frac{3}{4}$ -1-3-24 hours, usually three-quarter hour, the reading is made of the amount of sedimentation. The time (Linzenmeier, Frisch, Starlinger, etc.) method, tubes with two gradations, the lower one 18 m.m. from the upper are used, and the time necessary for the red cell column to drop to the 18 m.m. point is registered. In the graphic method, essentially a modification of the old distance method, readings are taken q 5 minutes, and recorded graphically.

Theory: The velocity sedimentation is a nonspecific biological reaction present whenever there is a marked protein destruction with resorption of the catabolic products entailed therein. The actual genesis of this reaction was disputed until investigation in the fields of capillary electricity and colloidal chemistry clarified the matter. Examination of the capillary circulation of a normal individual shows a uniformly finely granular field, whereas that of a pregnant woman or of patient suffering with an inflammatory illness, shows coarsely granular clumps. Fahreaus demonstrated these clumps in freshly prepared blood smears and claimed that these "new elements" were the cause of sedimentation. However, just as Hunter, by his ingenious experiments was able to place the cause in the blood plasma, so have many investigators been able to definitely place the cause in a change that occurred in the plasma which affected the electrical charge of the erythrocytes. Hober showed that the red cells wandered to the anode and were consequently negatively charged. Schurer and Eimer found that the red cells of healthy individuals migrated more rapidly to the anode than those of gravid women or sick patients, showing that the normal reds have a stronger negative charge. Linzenmeier, Hober, etc., found that the addition of proteins such as fibrinogen, protamin, histon, etc., caused a decrease in the negative charge of the red cells, with a concomitant accelerated sedimentation. It was further found that it was the globulin fraction of the fibrinogen that was active in producing this electrical change. Thus, in inflammations we have a diminution of the negative charge

due to an increase in the globulin in the plasma, which, by surrounding the red cells, causes the reds to clump. Then proportionate with increase of radii of clumps, they sediment more rapidly (Stokes law).

Clinical Application: Although Fahreaus introduced this test as a diagnosticum for pregnancy, it was found that its significance in that role was minor. Linzenmeier and others, however, applying it in gynecological conditions found it extremely useful. Here it served as an indicator as to whether operation should be undertaken in adnexal conditions. They apparently find it to be more sensitive than the temperature curve or the blood count in determining the transition of an acute salpingitis into a chronic stage. It also helps in differentiating uncomplicated ectopics from infectious processes in the same regions. In internal medicine it has also been found to be of definite value in the prognosis and treatment of pulmonary tuberculosis. Westergren, Starlinger and others consider the sedimentation test more valuable as an index of the course, value of treatment and fitness for discharge of patients suffering from tuberculosis, than other tests.

In the field of otology, however, this test has not been applied to any great extent. It was extremely interesting to determine whether this procedure might serve as an aid in the diagnosis, differential diagnosis and treatment of otological conditions. Could this test help differentiate an otitis media purulenta acuta from a mastoiditis acuta? Could it aid in foretelling the approach of complications in acute mastoids, postoperative?

With these questions in mind, I employed a modification of the Fahreaus test. A 2 c.c. graduated cylinder, divided into 100 equal parts, having an inner diameter of 4 m.m., was filled to upper mark with a mixture of 4 parts of blood to 1 part of sodium citrate (3 per cent). In a 2 c.c. dry syringe, or flushed with citrate, .4 c.c. of citrate is drawn, and then mixed with 1.6 c.c. blood obtained by venupuncture. Barrel is then drawn out a bit and the mixture turned up and down for one minute to insure thorough mixing. The mixture is then transferred into the cylinder alongside of a strip of capillary tubing to insure mixing and expedite filling. At the end of 45 minutes the reading is taken. This modification was employed as most practical, least time taking and most accurate. At the beginning of this series of tests, blood was taken daily, but as we progressed it was found that the results varied very slightly when taken that frequently, and that if these tests were taken every four or five days no information was sacrificed. Before any tests were started on the otological patients, 100 tests were done on apparently normal

individuals to determine the normal for the cylinder and method used. It was found that the normal for adult males was 3 to 5 per cent, adult females, 5 to 8 per cent, and children, 5 to 10 per cent.

For this preliminary report 75 otological cases were used. There were 400 tests done. Amongst the cases there were 30 acute mastoids, 6 subacute, 15 chronic, 1 meniere syndrome, 4 furuncles of the external auditory canal, 12 acute otitis media, and 7 chronic otitis media. Below I have summarized a few of the representative cases.

Case 1: I. S., male, age 68 years, admitted to the otological service on Nov. 9, 1926, complaining of purulent discharge from the right ear for the past four days.

Previous History: Incision of palmar abscess 30 years ago. Has been hospitalized for two months for diabetis mellitus, that he has had for the past two years.

Present Illness: Eight days ago patient took to bed with a sore throat. Next day he developed pain in the right ear, headache and fever. Discharge started four days before admission.

Physical Examination: Ostensibly negative except for local condition, which shows a perforation of right drum with escape of a blood-tinged purulent exudate.

Laboratory Data: Urine essentially negative; blood count, 4,100,000; hemoglobin, 80 per cent; leucocytes, 8,000; staff cells, 9 per cent; segmented, 43 per cent; and lymphocytes, 48 per cent. Blood chemistry on several occasions negative. Culture of aural discharge, nonhemolytic streptococcus. X-ray of the mastoids shows cloudy right mastoid cells, trabeculae hazy.

Operative Findings: Nov. 12, 1926, simple right mastoidectomy, large pneumatic mastoid in precoalescent stage, cells full of pus. Sinus not exposed, dura exposed over a small area found to be normal. Patient ran the usual postoperative course and, feeling well, was discharged Nov. 23. Returned, however Dec. 6, complaining of headache, almost continuous, vomiting a number of times. Wound still discharging pus. Revision of the mastoid revealed sinus leading from the surface by small canal into a large mastoid cavity full of pus. There were few gray stunted granulations, with bone over sigmoid sinus necrotic. Cavity was curretted, necrotic bone removed and wound packed.

Review of the chart and graph shows a persistence of the level of the sedimentation until the retain pus pocket had been evacuated. In this case it required two months time before it returned to normal.

Case 2: B. M., female, age 7½ years, admitted to the otological service on Nov. 23, 1926, with the complains of fever for nine days, pain and discharge from the left ear for six days.

Previous History: Measles, pertussus and chickenpox. Surgical tonsillectomy four years ago.

Present Illness: Took sick one week ago with a sore throat. A few days later she complained of pain in the left ear, which ruptured two days later and has been discharging ever since. For the past nine days she has been running temperature, sometimes as high as 104.8°.

Physical Examination: Negative except for local condition, which shows a marked purulent discharge from the left ear, with marked tenderness on pressure over the mastoid.

Laboratory Data: Temperature on admission was 102.6° and dropped gradually to normal. Urine negative. Blood counts varied from 3,200,000, hemoglobin, 65 per cent; leucocytes, 22,500; staff, 12 per cent; segmented, 55 per cent; mononuclears, 5 per cent, and lymphocytes, 26 per cent; to leucocytes, 9,000; staff, 4 per cent; segmented, 44 per cent, lymphocytes, 52 per cent on discharge. X-ray of mastoid showed slight clouding of the anterior region, with several large cells in the post-half of the mastoid.

This patient was watched and left the hospital without being explored. Chart and graph show definite speedy drop to the normal in two weeks.

Case 3: A. M., female, age 67 years, entered the service on Oct. 4, 1926, complaining of pain in the left ear for the past three weeks.

Previous History: Has had slight ringing and pain in the left ear for the past 20 years, without discharge. Has had frequent sore throats. *Present Illness:* About three weeks ago patient developed pain in the left ear of marked intensity. Consulted physician performed myringotomy. Since then there has been a thick, purulent discharge issuing from that ear.

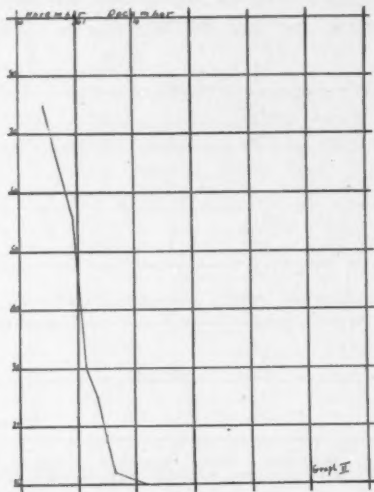
Physical Examination reveals slight tenderness over the left mastoid region, profuse, thick, purulent discharge from left ear.

Laboratory Data: Temperature during entire stay never rose above 100° until one week before exitus, when it became irregular between 98° and 101.4°. Urine on repeated tests showed presence of sugar, 1.5 per cent. Wassermann negative. Blood sugar on 15 tests varied between 170 and 390 m.g. Blood count varied from 9,900 to 30,000 leucocytes before death. Culture of discharge showed streptococcus mucosus. X-ray of mastoid showed moderate clouding of the cells of the left side, with marked absorption of the trabeculae.

Operation, Nov. 15, simple left mastoidectomy; coalescent type, thick pus throughout entire mastoid, granulation tissue in mastoid and over sinus. Patient, however, did not improve.

mittent type after mastoidectomy, culture positive, jugular ligated and sinus obliterated Sept. 27. Chills ceased, but temperature, in spite of intervals of normal temperature, rose to 105°. Patient was transfused with 300 c.c. of whole blood, direct method on Oct. 16. Following transfusion temperature started down and patient was discharged.

Graph and chart does not show the course of the entire illness as tests were started after the patient had been ill for one month. Transfusion given just as the sedimentation had reached its highest peak dropped materially and then in spite of a few interruptions

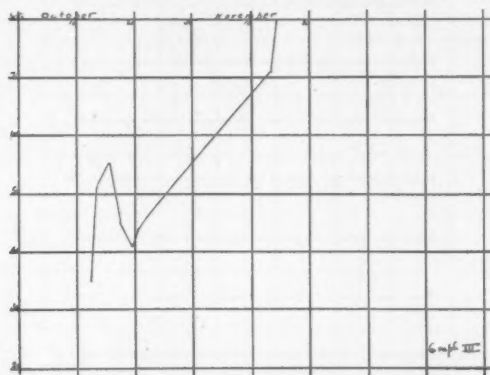


Graph 2. B. M. OMPA. Rapid drop to normal in seventeen days. Demonstrated absence of mastoid involvement. Patient discharged without operation.

returned to normal. In spite of the fact that the temperature disappeared immediately the sedimentation only improved gradually.

Comment: It was very striking to note that all chronic cases gave normal readings. Thus chronic otitis media, chronic mastoids with cholesteatoma or even labyrinthine symptoms, gave no increase results. Acute mastoids, and acute otitis media cases always gave a markedly accelerated sedimentation. The few cases of furunculosis of the external canal that I have seen gave no increase. We can thus gather that as far as diagnosis in otology the sedimentation test can play but a very minor role. I feel, however, that it might serve as an indicator in cases of acute otitis media where the question

whether the cleaning out of the mastoid cells should be undertaken. All of these cases should have sedimentation tests done at frequent intervals. If it is found that the results tend to remain high, or tend to rise that case should be prepared for operation. If the results show a tendency to drop, the operation can wait, and if the results return to normal no operation will be necessary. As a differential diagnostic sign between otitis media acute, acute mastoiditis and sinus thrombosis, I have found it to be of little help. However, as a prognostic sign and guide in treatment I believe it to be excellent. If, the tests being done serially, it is found that the level of the sedimentation is sustained or tends to rise, we can be sure that there is some complication intervening and that the infection is spreading. It also



Graph 3. A. M. Diabetes. Strep. Mucosus. OMPA—Mastoid. Died. Sedimentation test. Stayed high; rose to highest limit toward death.

informs the attending physician whether or not his treatment is efficacious. Contrary to the expectation that after operation the sedimentation findings would immediately drop to normal, I have found that it takes four to six weeks after operation before the results reach the normal level. Friedlander working with this test on pregnant women also found that instead of the sedimentation becoming normal after parturition, it generally took 10 days before it reached normal.

SUMMARY.

1. Normal amount of sedimentation for normal male is 3 to 5 per cent; of normal female, 5 to 8 per cent; and for children, 8 to 10 per cent. Acute mastoids, acute middle ear infections, sinus thrombosis, etc., runs values about 30 to 80 per cent. Subacute or

recurrent mastoids generally between 20 to 35 per cent, and chronic conditions between 2 to 7 per cent.

2. If done serially it may serve as an indicator for operations in cases of otitis media with questionable mastoid involvement.

3. Is of minor value in diagnosis, but is helpful as aid in prognosis and treatment.

CHARTS.

Chart 1: I. K. Diag. Diabetis-Mastoiditis, Cured.

11/10/26	57%	12/13	28%	12/30	10%
11/13	32	12/15	30	1/4/27	8
11/17	34	12/18	20	1/6	9
11/23	42	12/21	18	1/7	6
12/6	36	12/23	13		
12/11	12	12/27	15		

Chart 2: B. M. Diag. O. M. P. A. Cured.

11/24/26	75%	12/1	30%	12/6	12%
11/29	56	12/3	25	12/11	10

Chart 3: A. M. Diag. Diabetis, Strep. Muc. O. M. P. A. Mastoiditis.

10/13/26	35%	10/18	45%	11/14	71%
10/14	51	10/20	41	11/15	80
10/16	55	10/21	44		

Chart 4: S. G. Diag. Mastoid, Sinus Thrombosis, Cured.

10/14/26	50%	10/20	26%	11/7	8%
10/15	66	10/21	25	11/10	5
10/16	70	10/22	28	11/11	2
10/18	39	10/27	15	12/2	3
10/19	30	10/30	28		

Chart 5: Y. C. 3 years R. O. M. P. A., 30 per cent. A. M. 67 years Acute Mastoid, 80 per cent. C. C. 3 years Bil. O. M. P. A., 59 per cent. M. H. 20 years Chr. Mastoid, 4 per cent. M. P. 32 years Fur. Ext. Canal, 5 per cent. N. T. 25 years Cholesteatoma, 3 per cent. A. B. 38 years Meniere's, 6 per cent. M. Y. 32 years Recurrent Mastoid, 35 per cent.

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53 East 97th Street.

REPORT OF A CASE OF VASCULAR TUMOR OF THE MEMBRANA TYMPANI.*

DR. FRANCES RICHMAN, Brooklyn.

Through the kindness of Dr. Hans Brunner, I had the opportunity of observing the following case in the Ear Department of the Poliklinik Hospital in Vienna:

P. K., age 15 years; had no serious illness at any time. When seven months old, patient was operated on for some condition in the left preauricular region. About the same time there was a discharge from the left ear. According to the hospital report at that time, the patient had an acute otitis media, left, which apparently soon healed. However, since the age of seven months, patient has had poor hearing on the left side. Absence of tinnitus and labyrinthine dizziness.

Status Praesens: There is an asymmetry of the face, the left half being smaller than the right. There is a small, white scar about 1 c.m. anterior to the insertion of the left helix. The right drum



Fig. 1. Eleven-year-old girl. Hemangioma of left membrana tympani. It included the hiatus margin of upper quadrant.

picture is about normal. On the left, one can see three reddish-brown prominences in the posterior-superior quadrant, which cover the drum membrane to a large extent. The handle of the malleus is only partially visible, and the short process is entirely covered. Wherever the drum membrane is visible, we find it normal (Fig. 1).

On probing, these prominences feel like bullae covered with an impressible membrane. Incision of one of these bullae produces bleeding which is easily controlled by packing, but by the following day the vesicles are again tensely filled.

Accentuated whispered voice is heard on the left at 30 c.m., on the right at +12 metre; Weber not lateralized; Schwabach normal; watch heard from both mastoid processes; Rinné negative both sides;

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C₄ shortened about 10 seconds on the left, normal on the right. No spontaneous nystagmus; no Romberg. Caloric test: normal reaction both sides. Nose and throat negative. Eye examination: Myopia; fundus negative. X-ray of temporal bone: Highly irregular pneumatization; slight forward location of the lateral sinus.

The patient was referred to the radium station of the Allgemeine Krankenhaus, where she was given a course of radium treatments, the report of which follows:

Patient P. K. was irradiated: with a short Dominici tube, screen-age 0.3 m.m. platinum, sixfold layer of guttapercha as a secondary filter: On Sept. 22, 1926, 10 mg.h.; Sept. 29, 10 mg.h.; Oct. 13, 8 mg.h.; Oct. 27, 8 mg.h. With a barrel-shaped applicator having a radiation area of 4 m.m. diameter, containing 6 mg. radium, screen-age 0.3 m.m. platinum, sixfold layer of guttapercha as a secondary filter: On Nov. 24, 1926, 7 mg.h.; Dec. 7, 6 mg.h. The applicator was introduced into the external auditory meatus, as close as possible to the eardrum.



Fig. 2.

Following the treatment, further examination on Dec. 16, 1926, showed that instead of the blood vesicles, there was a tumor, the size of a small pea, resting on the drum membrane. On probing, the tumor feels hard and solid, and is movable, so that one has the impression of a sessile fibroma of the drum. The color of the tumor is gray; where it is covered with cerumen, it is black. The posterior-superior quadrant is now visible for the most part, and shows an atrophic scar and the long process of the incus. The handle of the malleus is also visible now (Fig. 2).

The hearing has increased markedly, so that patient now hears on the left accentuated whispered voice at 4-5 metre.

The case is very interesting, but complicated as regards a diagnosis. It was easy to recognize that there was no question of a myringitis bullosa in this case, since all acute accompanying symptoms were absent. The otoscopic picture suggested at once a vascular tumor of the drum membrane. However, tumors of this kind

are extremely rare. Marx has gone so far as to maintain in the *Handbuch der speziellen Pathologischen Anatomie und Histologie* von Henke und Lubarsch (bd. 12, 1926) that cases of isolated angioma of the drum membrane are not known for certain. The cases reported in the literature as angiomas are, according to Marx, vascular polyps. Up to the present time, the only completely examined case known is Fischer's (*Ztsch. f. Hals-Nasen-Ohrenhkl.*, Bd. 5, 1923, 221). In this case, there was a tumor projecting from the connective tissue layer of the membrana tympani, whose base covered the entire upper half of the handle of the malleus. The tumor consisted of a framework of connective tissue, throughout which were numerous large and small blood vessels, especially veins. The tumor was covered externally by the intact epidermis of the membrana tympani, and was limited internally by the normal mucous membrane layer of the drum. It is of further interest to note that this patient's auditory apparatus was abnormal in many other respects: Both Shrapnel's membrane and the pars tensa were abnormally long; the head of the malleus was of extraordinary size; the malleoincudal joint was deformed; the long process of the incus was divergent instead of parallel with the handle of the malleus; the stapes was longer than usual; the tegmen tympani consisted of two parallel lamellae of bone; and the tympanic cavity was exceptionally deep. Since there were neither signs nor remains of a recent inflammation in the middle ear, Fischer regarded this vascular tumor of the eardrum as a malformation in the sense of a faulty overproduction of blood vessels, and all the more so since it was possible to show other malformations in the middle ear. In spite of the contrary opinion of Wittmaack, who explains Fischer's vascular tumor as the residuum of a nipple-like prolapse of the mucous membrane, I agree with Fischer in his interpretation, and see in this case the proof that actual vascular tumors of the eardrum do exist, based on an abnormal development.

Our case undoubtedly has a certain resemblance to the histologically investigated case of Fischer's, in that: 1. the history of the illness in both goes back to infancy; 2. and this fact seems of especial significance—in our case the disease of the ear was combined with an asymmetrical development of the face and skull; 3. there was a highly irregular type of pneumatization of the temporal bone, which we likewise may be permitted to class as a sign of abnormal development. After considering all of these factors, we would be justified in pointing to the two cases as being analogous; were it not for the fact that in our case there is a history of acute otitis in infancy. Certainly at the time the patient came under our observation there was absolutely no sign of suppuration. However, since a histological

examination is out of the question, we are forced to acknowledge the inflammatory factor in the genesis of the drum tumor. We cannot consider the case one of highly vascularized granulation polyp, because of the fact that the tumor consisted of fairly large blood vesicles that were covered with a distinct membrane; further, that after shriveling of the tumor following the radium treatment, the area of the drum on which the tumor had been located appeared quite atrophic, but not perforated, showing that the growth came from the drum membrane, and not from the middle ear.

From these facts we draw the conclusion that in our case, as well as in Fischer's, an abnormal development of the vessels of the membrana tympani was the nucleus for the vascular tumor, and that, perhaps, the inflammatory condition of the ear in infancy was the exciting factor for the growth of the tumor.

Finally, let us refer to the excellent result the radium treatment produced in this case.

6072 Madison Street.

DOUBLE-END ARROW ELEVATOR FOR NASAL PLASTIC OPERATIONS AND SUBMUCOUS RESECTIONS.

DR. JAY N. FISHBEIN, Providence, R. I.

This elevator is arrow-shaped, made with one end having a fairly sharp cutting edge on all sides, and the other end blunt. It is designed to enable the operator to cut in all directions—forwards, backwards and laterally—without withdrawing the elevator or using force.

In plastic operations on the nose, especially where connective tissue binds the skin down firmly, the elevator can overcome this difficulty without undue trauma, by using it with a circular movement.



In submucous resections, difficulty is often encountered in separating the mucous membrane where ridges or spurs are present. Using the elevator carefully, the mucous membrane can often be more easily separated. It is especially useful where connective tissue is present and the mucous membrane does not strip easily.

The handle has been made octagonal to enable the operator to secure a firmer hold on the instrument.

178 Lippitt Street.

POST-AURICULAR EDEMA DUE TO ACUTE INFECTIONS.*

DR. ALFRED A. SCHWARTZ, New York City.

The appearance of edema over the mastoid process during the course of an acute suppurative otitis media frequently forecasts the necessity of surgical intervention. Other conditions may present themselves, however, which clinically resemble a "surgical mastoiditis", but where no suppurative process is present in the mastoid cells, and where surgery upon the mastoid is not indicated.

When, during the course of an acute suppurative otitis media, a postauricular swelling develops, accompanied by redness, tenderness over the antrum and mastoid tip, a sagging of the posterosuperior canal wall, and temperature, the diagnosis of a surgical mastoiditis is readily made. Frequently, however, one or more of these symptoms are absent, and then, when the postauricular edema appears, the diagnosis may be more difficult.

At times, a subperiosteal abscess due to suppurative mastoiditis drains itself through the antrum and middle ear, the mastoid resolves, and surgical intervention is unnecessary. Where, however, a positive diagnosis has been made, it seems poor judgment to wait for such a resolution to take place, as the sequelae may be serious; and even if operative measures are avoided, a chronic suppurative mastoiditis may persist, with its loss of hearing, otitic discharge, and the ever-present menace of intracranial complications.

When a subperiosteal abscess has drained itself, and then reforms, surgical intervention is immediately indicated.

Case 1: A. R., age 6 months, was seen by me on April 21, 1924. Two weeks previously a left acute suppurative otitis had developed, which ruptured spontaneously. After a few days, the discharge ceased, and a paracentesis was performed by the physician in charge of the case. One week later, the discharge again stopped, and a postauricular swelling, very tender to the touch, was noticed.

Examination on April 21 showed a moderate thick, purulent discharge in the left ear. There was a large postauricular swelling, very tender to touch, and pushing the ear forward. The temperature was normal and the general condition good. The child was sent to the

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Beth Israel Hospital, with the diagnosis of acute suppurative mastoiditis, with subperiosteal abscess.

The incision in the drum appeared inadequate and a paracentesis was performed immediately on reaching the hospital, and a profuse thick, purulent discharge ensued.

The swelling behind the left ear rapidly grew smaller, and in three days had disappeared. The discharge from the middle ear also cleared rapidly and after a stay of five days in the hospital the child was discharged.

After the paracentesis on the day of admission, the temperature was normal and remained at 98.6° throughout its entire stay at the hospital. He gained weight and looked perfectly well at all times.

An X-ray of the mastoids was taken, but, as was to be expected on account of the child's age, showed nothing definite.

On April 27, the day after discharge from the hospital, the patient was readmitted with a recurrence of the symptoms—a large postauricular swelling and only moderate thick discharge from the ear. The child's temperature was 102° , and there appeared sagging of the posterosuperior canal wall. The postauricular swelling was diffuse and not as large as on the previous admission.

A simple mastoidectomy was immediately performed. There was only a small amount of pus under the periosteum. A perforation of the cortex, one-quarter inch in diameter, was found. The mastoid process was quite large, and filled with pus and granulations. The sinus was exposed by the disease.

The temperature dropped to normal almost immediately after the operation and the patient made an uneventful recovery.

Comment: In this case, the diagnosis of suppurative mastoiditis, with cortical perforation was made on the first admission to the hospital. However, the age of the child, the general good condition, and the realization that there was no emergency caused us to delay operation, and the entire otitic picture cleared. When, however, the subperiosteal abscess recurred, the child was operated at once.

It is possible that the subperiosteal abscess might have cleared again, but two paracenteses had already been performed, and another was contraindicated. The pathological picture at operation showed that there would have been danger in delaying operation further.

A furuncle of the external canal wall complicating the suppurative otitis media may simulate the picture of an acute suppurative mastoiditis. In furunculosis, there is pain on tension of the auricle and the canal wall is exquisitely tender at the site of the furuncle. If, however, there is a diffuse external otitis, the diagnosis is obscured,

and the outstanding symptom is always the marked otitic pain, frequently so excruciating that it keeps the patient awake at night. This is accompanied by marked tenderness of the auricle to touch, and the appearance of a postauricular swelling does not greatly lessen the pain. Where a postauricular swelling is due to a suppurative mastoiditis, an amelioration of the otitic pain may be expected. The pain of mastoiditis is rarely as severe as that of a furuncle in the external canal.

In furunculosis, there is no temperature. In mastoidal infections there is usually some temperature, although it may be absent.

The appearance of the drum and the posterosuperior canal wall is of the greatest importance in arriving at a decision, and it must be borne in mind that an external otitis may cause the posterior canal wall to assume a position which seems pushed forward, and may even conceal the tympanic membrane.

When, during an acute exacerbation of a chronic purulent otitis media, a postauricular swelling appears, accompanied by a protrusion of the posterior canal wall, the diagnosis between a furuncle and a perforation through the posterior bony canal wall must be considered.

In cases of chronic supuration in the mastoid process, the discharge from the middle ear may not be profuse; or there may be a sudden diminution in the amount of discharge, with an increase of pain, and postauricular swelling. There is little or no temperature. Tenderness is usually not present over the mastoid process because of the sclerosis of the mastoid and because of the relief of tension in the mastoid process itself when the perforation has taken place.

In such cases operative measures should be immediately employed, a postauricular incision made, the perforation in the external canal wall searched for and the complete surgery indicated should be performed.

One case, a patient of Dr. S. J. Kopetzky, a boy, age 15 years, was seen with a large postauricular swelling, and a tender bulge in the canal. He had not felt ill at any time, and was brought in because of the protruding ear. There had been a slight otitic discharge for many years. At operation, the mastoid, except for a dense cortex, was found completely destroyed, and the sigmoid sinus exposed throughout its entire extent and occluded by an aseptic thrombus. The patient was operated by Dr. Kopetzky.

Another case, age 20 years, at the Gouverneur Hospital, presented symptoms clinically those of a furuncle of the canal. The patient was very comfortable and walking around, the only symptoms being

a large postauricular swelling, an exquisitely tender bulge in the external canal, and a slight aural discharge which had been present, unchanged in amount, for many years. The patient was in the hospital for a few days, and then it was decided to make an external incision. The fistula through the external canal wall was found, and the mastoid explored. The entire mastoid was destroyed, and a large temporosphenoidal abscess was evacuated. The patient died.

Not infrequently a postauricular lymph node becomes enlarged during the course of an acute suppurative otitis media, and the node is very tender. If the lymph node can be outlined, there is no difficulty in diagnosis. If, however, there is a surrounding edema, and the outline of the node is lost in the general swelling, the diagnosis is more difficult, and the appearance of the middle ear, the character of the discharge and the point as to whether the tenderness is deep, over the bone, or superficial as it is in lymph node involvement must be carefully studied.

P. E., age 5 years, was admitted to the Beth Israel Hospital on Feb. 22, 1927. Three months previously, the patient suffered from a general glandular enlargement, was acutely ill, and the diagnosis of "acute glandular fever" was made. At the same time, a brother presented the identical clinical picture. The disease lasted about five weeks and then cleared, and the child was well until five days before admission.

At this time, the glands of the neck again became enlarged, and a left acute suppurative otitis media developed, which ruptured spontaneously. On the following day, a right acute otitis media developed, which also ruptured spontaneously.

The child appeared acutely ill. There was a profuse, thin, purulent discharge from both ears, marked mastoid tenderness, marked tenderness over the glands of the neck, and the temperature ranged between 102° and 104°. The patient was then removed to the Beth Israel Hospital for observation.

On admission, the temperature was 101.8°. General physical examination, except for the otitic findings mentioned above, the adenitis, and a suppurative sinusitis (right antrum), was negative. The blood count showed a total of 27,500 white blood cells with a differential count of 82 per cent polynuclear leucocytes and 18 per cent mononuclears. The urine was negative. Culture from both ears showed a hemolytic streptococcus.

For five days after admission the temperature ranged between 100° and 104°, the pulse ranged between 112 and 130, and the respirations between 20 and 30. During this time there was a marked difference

of opinion as to the advisability of mastoidectomy. The child appeared acutely ill, there was a profuse discharge from both ears, especially the left, and marked mastoid tenderness. However, it seemed more probable that the enlarged lymph nodes were responsible for the temperature and clinical picture, and operation was deferred.

On Feb. 28, six days after admission to the hospital, the temperature dropped to normal and remained at 98° for four days. The left ear, which had appeared the worse, became dry; the lymph nodes became smaller and the child, except for a nasal discharge due to the sinusitis, and a slight discharge from the right ear, appeared entirely well.

On March 4, 11 days after admission, there was a rise in temperature to 100.6° , and for five days the temperature ranged between 99.6° and 102° . The left ear remained dry, although there was still some mastoid tenderness. The discharge from the right ear became more profuse, and appeared pulsating. Daily blood counts during this period showed a total white cell count of about 7,000, with a polynuclear count of 57 per cent. The red blood cells averaged 3,300,000, with a hemoglobin of 65 per cent. The cervical lymph nodes were again enlarged and tender.

On March 9, 16 days after admission to the hospital, a swelling appeared behind the right ear, pushing the auricle forward. Tenderness over the mastoid region was marked, and there was considerable pain. The discharge from the right ear was rather profuse. The child appeared very ill. The lymph nodes of the neck were large and tender.

In view of these clinical findings it was thought advisable to explore the mastoid, although the possibility of an enlarged lymph node causing the swelling was seriously considered. A simple mastoidectomy was performed. No perforation was found in the cortex, and no subperiosteal collection of pus was present. No pus was found in the mastoid, although granulations were found scattered throughout, especially in the region of the mastoid antrum.

The postoperative course was uneventful. There was a rise in temperature to 103° on the day following the operation, and the temperature ranged between 99° and 101° for a week longer. The wound healed rapidly and the adenitis gradually cleared.

Comment: The pathological findings at operation did not confirm the diagnosis of a surgical mastoiditis.

We feel now that a postauricular lymph node was enlarged, together with the general adenitis, and the edema about it caused the auricle to be pushed forward. This was suspected prior to opera-

tion, but the child appeared so ill, and the otitic suppuration had been so long continued that it was thought inadvisable to wait.

When the diagnosis of furuncle or lymph node involvement is considered in the presence of an acute otitis media, it should be borne in mind that operative measures upon the mastoid may be safely delayed, provided that the patient's general condition warrants such a course. For a postauricular swelling due to an acute mastoiditis usually means a perforation through the cortex, a decrease of tension in the mastoid, and an outlet for the pus under the periosteum. Such a delay would apply only in cases of acute suppurative otitis media; in an acute exacerbation of a chronic otitis, it is far safer to explore than procrastinate.

X-ray examination should be made in all such cases. This may not be of great value, for a clouding of the mastoid cells may be present, due to a nonsurgical mastoiditis, and the picture may be further obscured by the presence of pus over the mastoid, from whatever cause. A completely negative X-ray film of the mastoid, where the cells show a normal outline, is, of course, of the utmost importance.

A postauricular swelling which appears when there is no suppurative otitis may present problems for diagnosis.

When a postauricular lymph node is enlarged, the diagnosis is not difficult; for the tenderness is superficial and there is little or no fever. The tympanic membrane is normal.

An acute mastoiditis may occur without middle ear suppuration, and with the tympanic membrane intact; and the first visible sign of mastoid involvement may be a postauricular swelling due to perforation of the cortex and the formation of a subperiosteal abscess. There may be no pain or tenderness over the mastoid, and little fever. A simple edema at times occurs in this type of mastoid suppuration, where the mastoid emissary vein has been exposed by the disease there is a partial blocking of the venous flow from the scalp, through the emissary vein into the sigmoid sinus. As a rule, there is an accompanying suppurative otitis; but when the middle ear is dry, or normal, the problem is very difficult. Roentgen ray examination is of the utmost importance, and may be the deciding factor in the diagnosis.

There are types of postauricular edema without otitic suppuration which are more serious, and the differential diagnosis difficult. The following case is illustrative.

N. B., age 20 years, was first seen by me on Nov. 15, 1925. Three days before, he complained of pain behind the right ear, and over

the right parietal region, which persisted. These pains gradually became more severe, and he felt very feverish and complained of chilly sensations. A swelling appeared behind the right ear, which was gradually increasing in size and was tender to the touch.

When first seen, the patient appeared acutely ill, with a temperature of 104° , pulse 118, respirations of 32. There was present a slight swelling over the right mastoid region and marked tenderness over the mastoid and parietal regions. There was no redness of the skin.

The right drum was normal and hearing was unimpaired.

He was immediately sent to the Beth Israel Hospital for observation. A blood count showed a total leucocyte count of 10,900, with a differential count of 75 per cent polynuclear and 25 per cent mononuclear leucocytes. A blood culture was taken.

On admission to the hospital, the temperature was 104° , and dropped to 100° within 12 hours. There was no relief in the patient's condition; he still appeared prostrated, and complained bitterly of right-sided headaches. The temperature then suddenly rose to 105.4° ; he was chilly, but had no distinct chills. A slight redness developed over the postauricular swelling. Blood culture, 24 hours, was sterile.

Let us at this point summarize and consider the diagnosis.

Summary: The patient is acutely ill, with a septic temperature, complaining of severe headache, and appears prostrated. The only focal sign is the edema over the mastoid process. The middle ear is normal.

Diagnosis: There are three possibilities. First, a primary osteomyelitis of the temporal bone must be considered; or a mastoiditis, due to a middle ear infection which has cleared without suppuration. The temperature in these cases is usually low, rarely septic in type, and tenderness is deep, due to a periostitis; and the patient may appear ill, but is not prostrated. X-ray examination is very important, and may show definitely the location of the lesion.

The second possibility is a primary sinus thrombosis. There may be no suppuration in the middle ear or mastoid process, the infection having passed directly to the blood vessel, and the original infection in the middle ear or mastoid clearing without suppuration, and leaving the tympanum normal. The temperature is septic, rising to 105° and as suddenly dropping to normal. As a rule, there are distinct chills. Between these periods the patient is very comfortable, and the general appearance at such a time hardly coincides with the seriousness of the disease. Headache is an important symptom, but it

is not excruciating, not constant, and is frequently "neuralgic" in type. The edema behind the ear appears as a simple edema, pits easily, and is hardly tender to the touch.

Where such a diagnosis is probable, blood culture should immediately be made and often repeated. The presence of the hemophylic organisms in the blood stream indicate the sepsis.

When the diagnosis of septic sinus thrombosis is definitely made, it carries with it the obligation of immediate radical surgery to save the patient's life—and obliteration of the sigmoid sinus and ligation or resection of the jugular vein must be performed at once.

The third possibility to be considered is an erysipelas, which has first involved the postauricular region. The patient is prostrated, and complains of headache entirely out of proportion to the local symptoms presented. The temperature is septic, up to 105° and dropping suddenly to normal, although there may be a persistently high temperature. The patient always seems ill, even when the temperature is normal. The only localizing symptom is the edema of scalp, which is rather hard, and pits with more or less difficulty. There is little or no redness, and no line of demarcation until the disease spreads over the hair line. The region is markedly tender to the touch, but the tenderness appears superficial, and over any point where bone can be reached there is little or no pain on pressure.

The diagnosis of erysipelas immediately forbids the use of any surgery whatever—in contradistinction to septic sinus thrombosis—for operative measures may expose the patient to a virulent meningitis.

This type of patient must be hospitalized, and blood cultures taken immediately. If there is a positive culture, operative measures must be employed at once. If the edema spreads, and the diagnosis of erysipelas seems quite certain, and the blood culture is negative, the serum (erysipelas- streptococcus) should be used, and good results may be expected from it. The brauny look and elevated edges of the typical erysipelas usually do not appear until the seventh or eighth day, when it is seen over the forehead; and then continuing onward, may end as an edema over the opposite mastoid process.

Let us here bring the case of N. B. to its conclusion. For seven days, the temperature remained septic in type, its daily high point being between 105° and 106° and dropping to between 99° and 100°. On the eighth day the temperature dropped to 102° and then slowly subsided to normal.

The swelling behind the right ear gradually spread upward and forward to the forehead, leaving the mastoid region clear, and four

days after admission to the hospital redness and an elevated border made their appearance over the patients' forehead and the bridge of the nose, and confirmed the diagnosis of erysipelas. The swelling traveled rapidly to the left side of the head, and two days later there was an edema and slight redness over the left mastoid region. The temperature then dropped, and soon became normal.

Two blood cultures, one taken on the day of admission and one three days later, were sterile.

Several blood counts were made, averaging about 11,000 white blood cells with a differential count of 75 per cent polys. and 25 per cent monos. There was no anemia.

X-ray of the mastoids showed no destruction of the mastoid cells.

In presenting these types of cases briefly, it is to be noted that an edema over the mastoid region may be a symptom of various pathological conditions, which range from a furuncle to an erysipelas or a sinus thrombosis. The differential diagnosis is frequently very difficult and every medical aid, especially the laboratory, must be employed to arrive at definite conclusions.

203 West 90th Street.

FIRST INTERNATIONAL CONGRESS OF OTO-RHINO-LARYNGOLOGY.

The Congress will be held in Copenhagen from July 29 to Aug. 1, 1928, under the presidency of Professor Schmiegelow. The following subjects have been arranged for general discussion:

1. "The Modified Radical Treatment of Middle-ear Suppuration," introduced by Professor Tapia (Madrid) and Professor Neumann (Vienna).
2. "Septic Diseases Originating from the Throat," introduced by Professor Ferrari (Rome) and Dr. Uffenorde (Greifswald).
3. "Surgical Diathermy of Malignant Growths in the Upper Air Passages," introduced by Dr. Dan McKenzie (London) and Professor Gunnar Holmgren (Stockholm).
4. "The Anatomical Structure of the Ear and Its Influence On the Course of Suppuration of the Middle Ear," introduced by Professor Mouret (Montpellier) and Professor Wittmaack (Jena).

SEPTICEMIA SECONDARY TO ACUTE MIDDLE EAR INFECTION.

DR. CLAUDE G. CRANE, Brooklyn.

We are all agreed that an acute otitis media is never strictly limited to the cavity of the middle ear. There is always an invasion of the adjacent areas. This may only be the antrum and the adjacent mastoid cells, but more often the invasion is much more extensive. When the symptomatology is sufficiently mild, both locally and generally, that the patient is not ill, but is merely indisposed, we are accustomed to consider the situation as a simple otitis media. That there are many such cases that get well, with or without treatment, is only too obvious. We are not concerned in this discussion with this rather large group, but with all those cases that present the more serious picture of a local infection spreading to the adjacent areas and causing serious symptoms.

It is well to mention that the type of bacteria in our acute infections is highly important. A culture should be taken of every acute infection at the earliest possible moment. If we know the type of the invading organism we at once place ourselves in a position to make a decision in a doubtful case. The streptococci group, with few exceptions, produce our most virulent cases. The streptococcus capulatus mucosus (*Pneumococcus* Group III) is the most virulent, and at the same time the most insidious of the group. With this type of infection we are prepared to deal with a patient most critically ill and at the same time, with apparently little local pathology in evidence, very often on operation. The streptococcus hemolyticus is, I believe, the next most virulent type. It is more often than any other type found in thrombophlebitis of the lateral sinus and in the blood culture of the bacteremia cases. The hemolytic streptococcus is much more destructive in the blood stream than the vividans. That we may have in some cases transmutation of these types during the period of acute illness must be accepted.

Whenever we meet an acute infection of the ear that presents a picture of something more than the simple otitis media to which I have referred, it should become at once a hospital case. We cannot hope to properly diagnose and treat these severe cases unless we have them under our control in the hospital. The earlier such control is

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instituted the better the prognosis. On admission to the hospital, certain diagnostic measures should be immediately undertaken. I wish to emphasize this, as I believe we all have been guilty of delaying important and obviously routine examinations. A complete blood picture should be made at once, hemoglobin, red and white differential, a blood culture, and if the middle ear has not been cultured, it should be done at once. An X-ray of the mastoid area should be routine. The temperature, pulse and respiration should be q. 2 h. and specific instructions should be given to be watchful for the occurrence of even the slightest chilly sensation, as a distinct chill may never occur. The blood culture and the complete blood examination should be done daily. I will not say much about the frankly surgical mastoid. We all know what it is and what to do for it. I am concerned, however, with those cases that present, with or without an obviously surgical mastoid, a picture of a patient who is rather more ill than is to be expected from a simple otitis media, or even with a simple surgical mastoid. The patient may not be critically ill but sufficiently so to warrant our very careful scrutiny of all of our laboratory data, our clinical picture, and of every symptom which indicates that our local pathology is no longer a strictly local and walled off infection. Sleeplessness, irritability alternating with a sense of well being, dryness of the skin or periods of sweating, gastrointestinal disturbances, and any sign which gives us the impression that all is not well with our patient.

This, I realize, sounds like the bedside instructions we received as medical students. However, I have not always made an early diagnosis of otitic sepsis, far from it, and it has been because I have not always been watchful of the early signs and symptoms. I have not always given them the significance to which they were entitled. It is because I wish to make a plea for the early diagnosis of otitic sepsis that I emphasize these early and what often seem like insignificant symptoms. The blood picture may show a high leucocyte count and a high poly count, but oftentimes the reverse is true even with a severe sepsis. The high count merely means a combative reaction, while a low count means no response to the invading infection. The disappearance of the eosinophiles is significant of sepsis. The destruction of the erythrocytes and a low hemoglobin should be regarded seriously. The temperature curve is, of course, important. A persistently high temperature with or without remission, at regular or irregular intervals, with or without slight chilly sensations, or a distinct chill or sweating, always means something more than a localized infection. With such a picture presented, the mastoid should be operated, even though a frank surgical mastoid is not obviously pres-

ent. The X-ray may not show gross pathology. The ear culture may be the deciding evidence. When the mastoid is operated under such circumstances, the sinus should always be exposed and carefully examined, even though the sinus plate may on gross examination appear normal. The diagnosis of lateral sinus thrombophlebitis is not easy. If the pathology has progressed to the point of a well formed thrombus, it is obvious to the observer, but the early cases are not so easy but are most emphatically the most important. The early stages of a phlebitis cannot be diagnosed by any measures at our command. In the beginning, we have an invasion of the external wall of our sinus and then of the intima.

We may have a peripheral phlebitis of serious portent to the patient and still be unable to detect it. The blood stream is unimpeded. Such measures as aspiration are of no avail and should not be recommended. To my mind, it is these cases that should be treated decisively and promptly. To wait for a thrombus to form is courting disaster and lessening the therapeutic value of the obvious treatment. It is during the stage of peripheral phlebitis and before the thrombus forms that the active invasion of the blood takes place. This is the time for action. The formation of the clot is nature's attempt at a cure. That nature accomplishes a cure in this way is well recognized. The clot becomes sterile, the invasion of the blood stream ceases, the patient's blood takes care of the bacteremia and the patient gets well. The clot becomes organized, absorbed and obliteration of the sinus results. However, it is not our job to wait for this occasionally happy result. It is our job to stop the further invasion of the blood. We should not wait until we have the more serious conditions to deal with, such as secondary metastases. Some of these are more serious than the others. Endocarditis, osteomyelitis are nearly always hopeless. The joint invasions are not so serious. While we shall always avail ourselves of every measure to save these late cases, it is with the early cases that we are most concerned, because of our prospect of a happy result. In every case of sepsis, early or late, mild or virulent, our primary and, I believe, our most urgent and our most valuable single measure, is the ligation of the internal jugular. The lateral sinus should always be exposed. The ligature should always be done first, regardless of the apparent condition of the sinus. Even though you may believe you have not a phlebitis to deal with, in the presence of sepsis the jugular should be ligated. Whether you open your sinus or not, is, to my mind, of secondary importance. You have done the important thing when you have completed your ligation. You have shut off further invasion of the blood and, what is also of vast importance, you have

given your thrombophlebitic vein physiological rest, which is our most curative measure for the infected vein. The second most important therapeutic measure is blood transfusion. As soon as the first symptoms of sepsis appear, the patient should be typed and one or more donors matched, so as to be ready for your transfusion without undue delay. This should be routine. It is important that the hemoglobin and the erythrocytes should be charted every second or third day, as the indications for repeating the transfusions are the blood culture, the temperature and other evidence of continued sepsis, such as chills and the number of red cells and the hemaglobin. I might say here that I have not had any personal experience in the intravenous use of mercurochrome. From the reports I have read, I shall continue to rely on the above measures. The intravenous injection of antistreptococcus serum, to be used at the time of the transfusion, has not so far as I have used it, added to the above measures. It is obvious that we have a goodly number of cases of otitic sepsis due entirely to invasion of the blood stream through channels other than the sigmoid sinus. In children the floor of the middle ear may be very thin or absent, and direct invasion of the jugular bulb is invited. That we have often enough, a thrombophlebitis of the smaller veins in the temporal bones resulting in a bacteremia without gross evidence in the larger sinuses, I am well aware, and is another reason why we should not, in the presence of otitic sepsis, stress too much the apparently normal sigmoid sinus.

Before closing these remarks, I wish to say just a word about the difficult problem presented when we have an infection of both mastoids and an otitic sepsis or bacteremia. The problem is to decide which jugular should be ligated. Both sinuses should be exposed. If one is obviously pathologic, has a distinct thrombophlebitis, and the other is obviously not so involved, we have no problem. If, however, there is no gross difference in the two sinuses we have a difficult decision to make. With blood flowing through both sinuses it is obvious that compression of one or the other jugular veins will not tell us anything; that is, the pressure of the spinal fluid as measures by the mercurial manometer, will not be changed more with pressure on one side than on the other, as we have no obstruction in either sinus. This also holds true of the change in volume of blood of the veins of the forehead, etc. The aspiration with the needle of both sides, for the purpose of forming some judgment on the presence of a possible peripheral phlebitis or for the purpose of determining the presence of bacteria on one side or the other is also unreliable. It is possible, however, to tie off one jugular and a week later to tie off the other, as the interval allows the collateral circulation to be established. My only excuse for stressing early diagnosis in otitic

sepsis is the distinct impression I have had of the great number of cases reported in the literature in which the diagnosis has been made long after the onset of the systemic invasion. It has been estimated that thrombophlebitis occurs in about 4.5 per cent of all acute mastoid infections. The right sinus is larger and more susceptible to infection.

Some time ago, I read a report of a case which illustrates delayed diagnosis, even under what we are bound to regard as the most favorable circumstances for early diagnosis. The case was in one of the best hospitals of one of our great medical centers. The ablest specialists and internists were in daily consultation. The patient was admitted with a history of acute otitis media of three weeks duration. Examination on admission showed an acute otitis media. Every possible method of examination was made, but the clinical picture of sepsis was interpreted as typhoid fever for a period of nine days before the mastoid was operated and the thrombophlebitis revealed. This may seem to many of you a manifestation of lack of skill and experience on the part of those responsible for the delay, but I assure you that such was not the case. To go further afield, let me mention a case reported at the January, 1927, meeting of the Austrian Otological Society. The patient was admitted to the hospital in June, 1926, with a diagnosis of bilateral acute otitis media of 14 days duration on one side, and three days duration on the other. The patient was under observation for 10 days, at which time the temperature rose, accompanied with a rigor. Blood examination showed the presence of streptococcus brevis. Although, during the next four weeks, the blood continued to show a positive culture and a metastatic infection in one of the small joints developed, no operative procedure or any therapeutic measure whatever was attempted. The patient recovered. No comment on this case is necessary, but it is evident that some of our friends across the water believe in watchful waiting. So would we all, if we could be sure of such a happy outcome. Please do not interpret any comments I have made as a criticism of faulty diagnosis or unwarranted procrastination. My personal experience does not justify such an attitude. I have erred so often that I can fully appreciate the difficulties.

In reporting the following cases I will be as brief as possible, even at the expense of being incomplete.

Case 1: G. M., age 8 years, was first seen in consultation March 19, 1925. There was a history of acute otitis media of the right side of five weeks duration. For the last eight days the temperature had ranged from 104-105°, with delirium at night and irritability during the day. Examination showed a child profoundly ill. There was a profuse discharge from the middle ear. The only external evidence

of mastoid involvement was induration of the posterior edge of the tip. The patient was taken to the Brooklyn Hospital at once and operated immediately. A blood culture was taken before the operation, and on March 20 it showed a streptococcus hemolyticus. On operation pus was found at the knee of the sinus. The dura of the middle fossa and the lateral sinus were thoroughly exposed and carefully examined, but appeared to be normal. Following the operation the patient continued critically ill. Immediately after the operation 50 c.c. of antistreptococcus serum was given intramuscularly. On March 20, 1925, the spinal fluid was normal, although there were symptoms pointing to a beginning meningitis. On March 23, 1925, swelling in the region of the great trochanter on the left side and the shaft of the humerus on the right side was noticed. On March 24, 1925, these areas were operated for metastatic osteomyelitis. Culture from the osteomyelitis showed a short chain streptococcus. Transfusion was given immediately after this operation, 500 c.c. of blood being transfused. At the same time 100 c.c. of antistreptococcus serum was given intravenously. This patient returned from the operating room at 4 p. m. and died at 10:30 p. m. It is interesting to note the temperature continued high without any remissions. On admission it was 102° and ranged between 104° and 106° until the end. The cell count on March 20, 1925 was: leucocytes, 6,800; polymorphonuclear, 75 per cent; S. lymphocytes, 10 per cent; L. lymphocytes, 10 per cent; and on March 23 it was 20,800, 83 per cent, 15 per cent and 10 per cent, respectively. At the time of the mastoid operation I should have ligated the jugular and given a blood transfusion. This was the proper procedure and I believe it would have given this patient the only chance there was of surviving. This would have been on March 19, and the osteomyelitis did not manifest itself until March 23, so we would have been at least two days ahead of the metastatic development. This was a desperate case, but it illustrates some of our points unusually well.

Case 2: E. C., age 23 years, was admitted to the Brooklyn Hospital Dec. 11, 1925. *Diagnosis:* Chronic mastoiditis. Lateral sinus thrombophlebitis bacteremia. On the night of Dec. 13, he had his first distinct chill and the temperature rose to 105°. On Dec. 14, the mastoid was operated and the sinus was found to be thrombosed. The jugular was ligated, the thrombus removed and a transfusion was done two hours later. A blood culture taken before operating showed the streptococcus vividans. The blood became sterile on Dec. 23. The convalescence was satisfactory. Two more blood transfusions were done to maintain a satisfactory hemoglobin and erythrocyts count. He was discharged from the hospital on Jan. 9, 1926, apparently well. He was again admitted on Feb. 12, 1926.

The tentative diagnosis was encephalitis, meningitis, or possible brain abscess. The blood culture at this time was sterile. The spinal fluid was negative. The neurologist favored a localized meningitis and encephalitis. No localized symptoms were elicited. He died Feb. 20, 1926. Postmortem revealed a large abscess cavity in the left lobe of the cerebellum and included the fourth ventricle. It was evidently an old abscess and was believed to be of metastatic origin.

A period of 68 days ensued from the date of the operation on the sinus to death from the abscess.

Case 3: J. H. W., age 54 years, was admitted to the Brooklyn Hospital Jan. 25, 1927. He gave a history of having had an acute otitis media for four weeks. During the four weeks he was said to have had a slight fever at times and some sweating and chilly sensations. Examination on admission revealed a sinus in the mastoid region, which was discharging pus. The temperature, pulse and respiration were normal and the patient was free of symptoms. The blood examination was normal. Operation revealed a completely infected sinus with numerous perforations and a free discharge of pus. The jugular was ligated and the sinus cleared from the bulb to the torcular. There was also a large epidural and perisinus abscess. The blood culture was sterile. He was discharged on the seventeenth day, and complete recovery followed.

Case 4: L. I., age 10 years, was admitted to the Brooklyn Hospital June 26, 1927. On admission patient was found to be critically ill, with a high temperature and septic appearance. There was a history of acute otitis media of the left side of 10 days duration. During the last four days he had vomited several times each day. There was some tenderness over the tip of the mastoid, but no swelling. There was definite rigidity and tenderness over the right side of the abdomen. The X-ray showed no distinct cells on either side. The right side showed O. M. P. C. Medical examination was negative. Surgical examination showed marked rigidity and tenderness of right upper quadrant and left kidney region. Suggested possibilities—left pyelitis, right chest lesion, diaphragmatic pleurisy or central lung infection, also colitis and subhepatic or subdiaphragmatic abscess of metastatic origin. Neurological examination was negative. The urine showed albumin plus, and red and white blood cells. The blood examination: June 27, 1927, leucocytes, 24,200; polymorphonuclears, 92 per cent; S. lymphocytes, 7 per cent. July 2, 1927, hemoglobin, 68 per cent; erythrocytes, 3,410,000; leucocytes, 38,200; polymorphonuclears, 92 per cent; L. lymphocytes, 8 per cent. July 7, 1927, hemoglobin, 55 per cent; erythrocytes, 3,590,000; leucocytes, 15,000; polymorphonuclears, 80 per cent; L. lymphocytes, 20 per cent. Pus from the mastoid, June 27, 1927, showed long chain streptococcus and

short chain streptococcus. Blood culture, June 26, 1927, showed hemolytic streptococcus; and on July 1, 1927, it was sterile. The temperature on admission was 105.2°, pulse 124, respiration 32. Following operation it gradually subsided to normal, 14 days after operation.

The mastoid was operated on June 27, 1927, and a perisinus abscess was found. The sinus was soft and pulsating. The jugular was ligated. Examination of sinus on July 2, 1927, showed it to be soft and fluctuating and covered with healthy granulation tissue. Discharged July 14, 1927, and has continued well. The only measures used to combat the sepsis were the mastoid operation and the jugular ligation.

Case 5: J. K., age 70 years, was admitted to the Brooklyn Hospital April 10, 1927. He gave a history of having had a discharge from the left ear for 10 months, with pain behind the ear for two weeks, sufficiently severe to cause sleepless nights. The discharge had at times been copious. Examination on admission showed otitis media with tenderness of tip of mastoid. The heart examination showed very feeble heart sounds, giving a distinct impression of a very poor heart muscle. The general appearance was that of a very feeble old man who had been ill for some time. The urine examination was negative. Blood examination: April 11, 1927, leucocytes, 7,000; polymorphonuclears, 77 per cent; S. lymphocytes, 23 per cent. April 20, leucocytes, 21,200; polymorphonuclears, 94 per cent; S. lymphocytes, 6 per cent. Blood culture, April 19, 1927, showed hemolytic streptococcus. The temperature on admission was 101°, and dropped to normal two days after operation.

The left mastoid was operated April 11, 1927. The mastoid was found to be densely sclerotic, except a small area near the antrum. The sinus was exposed and appeared to be normal. The postoperative condition was normal until the third day, when he complained of pain in the left ankle and the posterior surface of the right knee. Examination revealed no swelling but appeared to be tender. On April 15, four days postoperative, the temperature rose to 103° in the evening. On April 19, swelling, with fluid in the right knee was in evidence. From this time on, he had a septic temperature and sweating. On April 21, hiccough developed. On April 22, he became steadily worse. The ligation of the jugular was proposed as soon as evidence of sepsis was present. The family did not support the proposal, but on April 24 I succeeded in receiving consent. As the patient was being prepared to go to the operating room, he very suddenly died. The positive blood culture on April 19 was a positive indication for ligation of the jugular, but of course it should have been done on April 14, at which time his temperature began to rise

and he first complained of pain in the ankle and knee. He was an old man, feeble and a poor risk, but the ligation at this period might have saved him. Blood transfusion at this same period was also indicated.

Case 6: M. M., age 34 years, admitted to the Brooklyn Hospital May 20, 1927. Three days before admission to the hospital had pain, followed by discharge from the right ear. Pain in the left ear was also complained of. Examination revealed a copious discharge from the right ear and mastoid tenderness. The left ear showed congestion of the drum. On the same day the right mastoid was operated. The mastoid showed free pus and blood clots. Culture taken. The urine showed, on May 20, albumin, 1 plus. The blood examination, on May 26, was leucocytes, 13,700; polymorphnuclears, 74 per cent; S. lymphocytes, 22 per cent. On June 9, the hemoglobin was 70 per cent; color index, 9; erythrocytes, 4,310,000; leucocytes, 15,200; polymorphonuclears, 81 per cent; S. lymphocytes, 14 per cent. On June 14, three days after transfusion, the hemoglobin was 70 per cent; erythrocytes, 5,480,000; leucocytes, 11,000; polymorphonuclears, 78 per cent. On May 24, the left drum was incised. The pus from the mastoid showed a long chain streptococcus. The blood culture taken showed, on May 25, a hemolytic streptococcus. The blood became sterile on June 3, and remained so. On June 11, a blood transfusion was done as a therapeutic measure, although the blood had become sterile and the temperature normal. The temperature on admission was 101° and for three days postoperative it was 102.2°, but gradually subsided and became normal 11 days after operation.

Here was a patient with a hemolytic streptococcus in the blood stream, who had no symptoms of sepsis and who had no treatment except the usual mastoid operation. Undoubtedly many patients with bacteremia get well without our even knowing they had a bacteremia.

Case 7: M. T., age 9 years, was admitted to the Brooklyn Hospital Aug. 3, 1927. Five days previous to admission, both ears began to discharge, following an acute respiratory attack. Temperature for three days before admission had ranged from 101-104°. On admission the temperature was 103.6°, pulse 124, respiration 20. The child was said to have lost considerable weight during the last few days. Examination on admission showed a child who appeared to be only moderately ill. Both drums were bulging. There was some tenderness over the tip and antrum of right mastoid. Paracentesis of both drums was done at once, and X-ray taken of mastoid. The X-ray showed only very slight haziness of the cells of both mastoids, and was not considered pathologic. The temperature on admission was 103.8°, and soon subsided to normal on third day. On the fifth

day it rose to 102° , and on the seventh to 104° , and continued high, with only slight remissions, until Aug. 22. It again rose on Aug. 27 to 102° and subsided to normal Aug. 30, and so continued until discharged Sept. 7, 1927. The blood culture on Aug. 13, 1927, showed a hemolytic streptococcus. On the same day the left mastoid was operated. The sinus was exposed and examined and appeared to be normal. Blood transfusion was given four hours after the operation, 250 c.c. of blood being given. On Aug. 16, the right mastoid was operated. The sinus was exposed and examined and appeared to be normal. Two hours after the operation, 200 c.c. of blood was transfused. The blood picture was as follows: Aug. 3, 1927, leucocytes, 16,100; polymorphonuclears, 92 per cent; S. lymphocytes, 8 per cent. Aug. 11, leucocytes, 13,200; polymorphonuclears, 78 per cent; S. lymphocytes, 22 per cent. Aug. 15, leucocytes, 13,000; polymorphonuclears, 88 per cent; S. lymphocytes, 12 per cent; hemoglobin, 65 per cent. Aug. 16, hemoglobin, 65 per cent; color index, 7; erythrocytes, 4,600,000; leucocytes, 13,000; polymorphonuclears, 88 per cent; S. lymphocytes, 12 per cent. Aug. 19, hemoglobin, 70 per cent; color index, 2.68; erythrocytes, 5,120,000; leucocytes, 12,400; polymorphonuclears, 71 per cent; S. lymphocytes, 29 per cent. Aug. 22, hemoglobin, 55 per cent; color index, .48; erythrocytes, 5,884,000; leucocytes, 13,600; polymorphonuclears, 52 per cent; S. lymphocytes, 48 per cent. Aug. 24, hemoglobin, 50 per cent; erythrocytes, 4,960,000; leucocytes, 13,900; polymorphonuclears, 60 per cent; S. lymphocytes, 20 per cent. Aug. 30, hemoglobin, 40 per cent; erythrocytes, 4,224,000; leucocytes, 10,600; polymorphonuclears, 61 per cent; S. lymphocytes, 39 per cent. Sept. 1, hemoglobin, 60 per cent; erythrocytes, 4,600,000; leucocytes, 10,100; polymorphonuclears, 64 per cent. The blood became sterile Aug. 29. This patient continued perfectly well, but a sinus of the right mastoid persisted, and the patient was admitted to the hospital, and on Oct. 24 the mastoid area was explored. No bone pathology was found. The lateral sinus was found to be solid, a large part of the wall was absent. The thrombosis had apparently become organized and complete obliteration had occurred. As this patient had been perfectly well since Sept. 7, it was decided that nature had effected a satisfactory cure and operative interference was not indicated. The ligation of the jugular was considered and believed not indicated. At the time the blood culture showed a hemolytic streptococcus the problem of which jugular to ligate was gone into, and it was decided not to ligate either one. At that time it seemed that the left side was the more likely offender, but later it proved to be the right side.

353 Washington Avenue.

SNARE-DISSECTOR TONSIL OPERATION.

DR. WILLIAM LAWRENCE GATEWOOD, New York.

All tonsillectomy operations in this country have been based, during the last ten years, on the fundamentals of two methods—the Sluder, or Beck-Sluder, and the “dissection and snare”. All new methods of procedure, including the one described below, must of necessity be but modifications of these basic methods with a view towards improvement.

While teaching the postgraduate students at the Polyclinic Hospital, we were impressed with the difficulties and defects of these methods. The shortcomings were particularly accentuated by the beginner, as an expert can adapt himself readily to any method or instrument.

In the Sluder, or Beck-Sluder, method the objections were: 1. the considerable amount of force and pressure necessary, causing marked discomfort in a local operation and subsequent marked tissue reaction; 2. lack of opportunity for direct vision, due to comparatively bulky instrument in small space.

In the pure dissection method: 1. the separator, whether knife or scissors, frequently straying from the path of cleavage of the capsule into the muscular adjacent tissue; thus causing subsequent adhesions, contractures and other damage; 2. the use of an additional instrument to dissect, thus complicating and lengthening the operation.

We have been led, by certain factors, to undertake the simplification of this operation. We have observed: *a.* that the posterior pillar is usually not very adherent to the tonsil in the average case; and *b.* that a great number of infant tonsillectomies can be performed expertly by the simple use of a tonsil-grasping forceps and a snare with wire arranged as described below.

For the last two years, in a large number of cases, we have used the cannula of the snare to push the anterior pillar outward from the capsule, thus aiding the release of the capsule. The grasping forceps lifts the tonsil away from the posterior pillar, while the oval-shaped wire engages the capsule in front of the posterior pillar.

Description of the Instrument: The particular features of this instrument are the modified shape of the cannula and the new arrangement of the wire. The distal end of the cannula is flattened and

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widened into a crescent shape. The two tips of this crescent-shaped end form two prongs. The distance between the two prongs, which are notched for the reception of the wire, is 14.3 m.m. This flattened cannula is the separator. The wire is oval-shaped to conform to the shape of the tonsil. The greater axis of this oval runs longitudinally with and parallel to the pillars when applied.

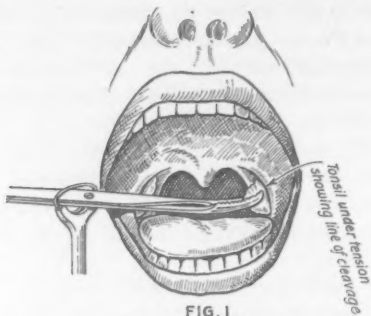


FIG. 1
STEP 1—Tonsil (engaged by forceps) in tension.



FIG. 2
STEP 2—Cannula separating Tonsil from Anterior Pillar.

The Operation: Through the loop of the snare the tonsil is grasped with an 8-inch-long curved, mouse-tooth, scissor-handled forceps, whose blades are about $1\frac{1}{2}$ inches long. The grasp is made just behind the anterior pillar and as much of the tonsil as can be grasped is included in the bite. The upper blade is fixed first by firm pressure into the capsule, just below the superior angle of the converging pillars, and the lower jaw of the forceps is then pressed to the same depth, seizing the inferior reflexion of the capsule, sufficient pressure

being used to prevent dislodgment of the blades before locking the handles. If this maneuver is properly performed, the capsule will be included in the bite. This is the most difficult and most important step in the operation, and its correct performance is essential to the smooth and rapid completion of the enucleation. When the tonsil is buried the blades are introduced in apposition between the pillars and are then separated so as to release the tonsil, and the procedure above described is carried out.

The left tonsil is grasped with the forceps in the left hand, the right with the forceps in the right hand. The forceps are then drawn



FIG. 3

STEP 3- Anterior Pillar pushed aside and instrument in position for constriction of wire, thus completing the operation.



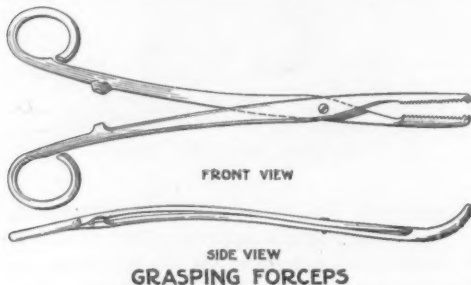
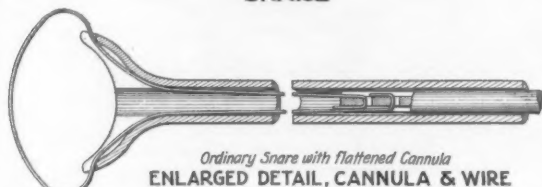
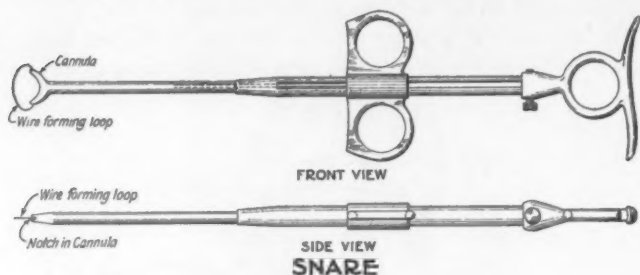
FIG. 4

Diagrammatic View of Cannula and wire engaging Tonsil.

inward and rotated so that the convexity is inferior and they serve as a tongue depressor, which is now discarded. The solid grasp and pull on the tonsil puts on tension the fibrous elastic and muscular tissues of the capsule. Great care should be taken not to include the pillars in the grasp.

The pull on the tonsil delineates the edge of the anterior pillar. The snare is then adjusted so that the posterior part of the oval-shaped wire is placed close to the posterior pillar of the tonsil. The oval lip of the cannula-separator is placed at the edge of the anterior pillar and insinuated between it and the capsule of the tonsil. It is essential to see the glistening capsule before proceeding further. The

mucous surface of the anterior pillar is undisturbed. Comparatively speaking, very little force is used to push the anterior pillar away from the capsule. The cannula-separator dislodges the tonsil from behind the anterior pillar, sliding the anterior pillar and its covering membrane away from the capsule of the tonsil. The operator has the sensation of peeling from the capsule. We must stress the fact that very little separation of the anterior pillar is needed, and none



at all of the posterior pillar. The anterior margin of the capsule is now brought into view. The operation should be quickly completed at this point by contracting the oval-shaped wire of the snare.

There is no purse-string action of the snare, which usually tears the membrane away or removes some of the posterior pillar. Just prior to bringing the stilet of the snare home, the handle of the instrument is carried outward, which position favors easier comple-

tion of this manipulation. The snare wire arranged this way gives all the advantages of the Sluder, or Beck-Sluder instruments, cutting from before, backward, besides allowing greater flexibility, thus permitting its insinuation behind the tonsil. The wire follows a line of cleavage of the plane of the capsule that contains blood vessels of most minute calibre.

Very little force is used during this procedure, and we impress the students with the need of gentleness in handling the tissues. The operation should not require a longer time than one minute per side. The condition of the tonsillar fossa is inspected by retracting the anterior pillar. An oval-shaped, tightly packed gauze sponge is wedged into the fossa. A string is attached to it, thus facilitating its removal. The bleeding, if any, from the side first operated on, usually the right, should have ceased before attacking its fellow.

On the subject of tonsillar bleeding, we take the position against too much meddlesome interference. If there is distinct bleeding from a blood vessel that does not subside by pressure, we put on a clamp for a minute or two. If the bleeding still persists we ligate and tie. While a definite bleeding point must be surgically controlled, we have noticed that the attempt to unnecessarily clamp and ligate causes needless traumatism.

The points of this instrument and operation are that only two instruments are used: the grasping forceps and the snare, the widened cannula of which acts as a separator; that very little separation of the anterior pillar is needed, the particular shaped cannula-dissector cleaving its way between the anterior pillar and the tonsil during the operation; that the posterior pillar does not need to be separated, owing to the oval shape of the wire.

No instrument or operation can be perfect, and certainly no such claim is made for this instrument and operation; but we have found that students grasp it very readily and easily get accustomed to it.

The operation has the advantages of: *a.* rapidity with due regard to safety; *b.* minimum amount of traumatism of surrounding tissue; *c.* the simplicity of the manipulations in the operation and the use of only two instruments; *d.* least possible amount of hemorrhage; *e.* suitability to the greatest number of operations.

The end results of the operation are particularly impressive as to the sharp outlines of the edges of the pillars. The tonsillar fossa is clear. There are no tears in the superior constrictor muscle. On inspection in three or four days, there is a minimum amount of reaction.

330 Park Avenue.

**A NEW SPHENOIDAL TROCHAR AND CANNULA FOR
DIAGNOSTIC PUNCTURE AND TREATMENT
OF THE SPHENOIDAL SINUS.
TECHNIC.***

DR. WILLIAM SPIELBERG, New York City.

During the past two years I have experimented with several types of instruments for puncturing the sphenoid sinus, either for diagnostic or therapeutic purposes. None, however, were found to answer the purpose from a satisfactory viewpoint, for the following reasons:

1. All were in form of thin, fragile appearing needles with or without safety devices.
2. The calibre of these needles was found to be too fine, particularly when used for a diagnostic puncture. Thick pus, mucoid or gelatinous matter could not flow through it.

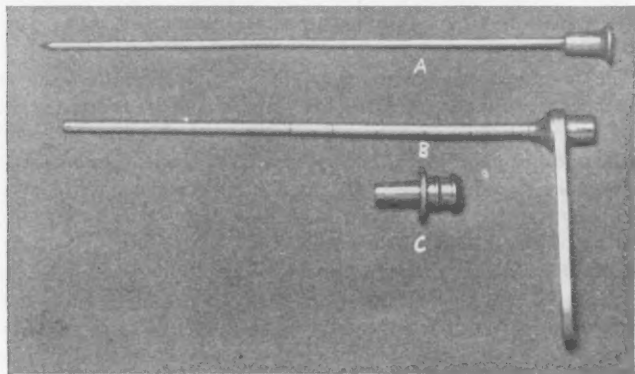


Fig. 1.

3. Neither could they be used for enlarging the puncture made, as they would break very easily.

The trochar and cannula here described was constructed on the same principal as that of the straight trochar and cannula used by me in puncturing and antroscoping the antrum of Highmore. It meets all the requirements necessary for approach to the sphenoidal sinus and has none of the disadvantages of the other instruments used.

The instrument consists of a trochar and cannula (Fig. 1).

*From the Otolaryngological Department of the Beth Israel Hospital.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Sept. 27, 1927.

The cannula (Fig. 1B) is straight and of uniform diameter in its entire length. It is graduated in centimeters. The first mark is 5 c.m. from the distal end. It has an inner diameter of 2 m.m. and an outer of 3 m.m. The point of the trochar (Fig. 1A) projects 3 m.m. out of the distal end of the cannula.

The instrument is provided with an obturator (C, Fig. 1) for irrigating the sinus.

Technic: The sphenoidal sinus should always be punctured or operated upon under control of direct vision. An X-ray plate show-



Fig. 2. Right. X-ray plate showing trochar and cannula 1½ c.m. within the sphenoid sinus. The latter was punctured at the time the X-ray was taken in the laboratory of Dr. L. J. Friedman, who X-rayed the patient.

ing a lateral view of the sinus should always be before the operator during the operation.

1. The nasal chamber is thoroughly swabbed with a solution of 10 per cent cocain and adrenalin equal parts, in order to shrink the inferior and middle turbinates.

2. An extra long-bladed Killian speculum is inserted into the nose between the septum and the middle turbinate. The blades are forcibly separated thus pushing the middle turbinate tightly up against the outer nasal wall.

3. The anterior wall of the sphenoid is thus brought into view and is thoroughly swabbed with the cocain-adrenalin solution. The natural opening of the sphenoid can now be located and probed.

4. With the long Killian speculum in situ or replaced by a small nasal speculum, the sphenoidal trochar and cannula is inserted along the floor of the nose until it impinges on the nasopharyngeal wall. Raising the distal end of the instrument to an angle of 45 degrees brings it to a point on the anterior sphenoidal wall most suitable for puncturing the sinus.

5. The handle of the instrument is now raised to a horizontal position and, directing it downward and backward, the anterior wall is punctured. The cannula is allowed to penetrate into the sinus for 1 to 2 c.m., or as much as may be required by the case. The operator is guided by the X-ray plate before him.

6. The trochar is then removed and the cavity investigated. A sterile culture can be very easily obtained through the cannula. The opening can be enlarged by first replacing the trochar and then by a rocking motion break up the anterior portion of wall about the puncture. The opening can subsequently be still more enlarged with a biting forceps. The sinus can be irrigated by inserting the obturator into the cannula.

COMMENT AND CONCLUSIONS.

It is of importance to emphasize the fact that a most thorough knowledge of the anatomy of all the intranasal structures is most essential before attempting any operation on the sinuses in general and the sphenoidal sinus in particular.

From an exhaustive study of the sphenoid sinus in many cadavers, including measurements taken from the latter as well as from patients from my private and hospital practice, I am lead to the following conclusions:

1. The sphenoid sinus is a large cavity, easily accessible both to diagnostic puncture and surgical treatment.

2. It can be approached without any greater danger or risk than any of the other nasal sinuses. Next to the antrum of Highmore I consider the sphenoid easiest to puncture or uncap.

3. A trochar and cannula is safest and most suitable for puncturing the sphenoid sinus, and that in the hands of the experienced no safety devices are necessary and are, in fact, a hindrance.

4. Sinus needles have been found inadequate and dangerous.

5. From our experience with the instrument herein described, we find it most suitable and safe for puncturing, irrigating and treating the sphenoid sinus, both in adults and children.

211 Henry Street.

THE BOECKMAN-KAPLAN SAFETY ADENOID CURETTE.*

DR. CARL KAPLAN, Brooklyn.

In the operation of adenoidectomy the aim and effort of all nose and throat surgeons is to remove completely all of the adenoid tissue in the nasopharyngeal space. Many of us feel very much embarrassed to see some of our cases return with symptoms of recurrence after we thought we performed a complete, satisfactory operation for their removal. These recurrences are due to small pieces of adenoid tissue left in the recess of the posterior wall of the epipharynx or in the fossae of Rosenmüller.

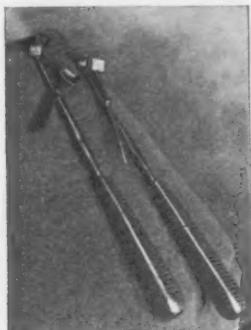
The most popular method of removal of adenoids today is the LaForce adenotome; however, after one has operated on several hundred cases with this method and has made a digital examination of the nasopharynx after each operation, he is surprised and amazed to learn how much adenoid tissue is still present in the spaces mentioned above which cannot be reached with the smallest LaForce adenotome.

These tabs are visualized with the digital finger and are usually located in the superior posterior wall of the epipharynx or laterally near the fossae of Rosenmüller. The tabs must be removed or we will surely have recurrence of adenoids.

The question now looms upon the best, safest and most thorough manner and method of removing these tabs. Today when such a phobia exists for post-tonsillectomy and adenoidectomy lung abscesses we must be careful in removing these tabs so that they are not aspirated into the tracheobronchial tree and thus produce toxic absorption, or even lung suppuration.

The ordinary curette is not safe, because we curette and scrape away adenoids. Part of these adenoid tabs are attached to the curette and removed through the mouth. The greater portion of these scrapings, however, are lost or left in the nasopharyngeal space and may find their way down the pharynx through the larynx and into the tracheobronchial tree. Why is it not possible that these small tabs of adenoid tissue aspirated into the lungs are not responsible for a great

*Presented before the Otolaryngological Section of the Kings County Medical Society.



Boeckman-Kaplan safety adenoid curette.



Boeckman-Kaplan safety adenoid curette. (A) Adult size. (B) Infant size.



Posterior view.

number of lung abscesses following tonsillectomy and adenoidectomy operations?

In view of this possibility we thought that an adenoid curette should be so constructed as to make it safe for the removal of these tabs of adenoid tissue. Therefore, I take the privilege of presenting for your approval a new safety adenoid curette. It is the old Boeckman curette made safe by building around it a small curved, closed chamber. Anteriorly it is fitted with a small closed spring door which can be opened and the small pieces of adenoid tissue removed. The curette is small and curved. This was made in such a manner so as to enable the operator to carry the instrument into any part of the nasopharynx and into the various recesses and the fossae of Rosenmüller.

There is no danger of any of these adenoid tabs becoming lost in the epipharynx, because as the adenoid tab is removed, it drops into the closed chamber below; upon removal, the door is opened and we can see all the adenoid tissue removed. This instrument should only be used after the greater portion of adenoid tissue has been removed with the LaForce adenotome.

I have used this safety curette since July, 1926, in many cases and I found it very satisfactory, efficient and easy to operate. The instrument is made in two sizes: *a.* adult; *b.* child.

I am greatly indebted to Pfau and Company, New York and Berlin, for their help and co-operation in the construction of this safety adenoid curette. They are the sole manufacturers.

I wish also to express my thanks to my colleagues, especially to Dr. Ed. Leo Berger for his suggestions and moral support.

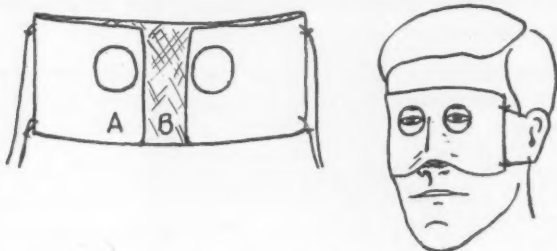
967 Forest Avenue.

ADHESIVE PLASTER BANDAGE FOR PLASTIC NASAL OPERATIONS.

DR. JAY N. FISHBEIN, Providence, R. I.

The purpose of this bandage is to exert pressure on the nose following plastic operations, without obstructing or covering the eyes. Edema is an important factor and comes on rapidly following these operations, which the bandage tends to control to a certain extent. Dental compound is used in conjunction with the bandage. The compound is warmed and moulded on the nose, and then fixed by cooling. The adhesive plaster bandage is placed over it and gives an even, firm pressure without interfering with the vision. It is easily made and applied, and inexpensive.

The bandage is made by taking a strip of adhesive $16\frac{1}{2}$ inches by $3\frac{1}{2}$ inches. Both ends are brought back on themselves, bringing the



(A) Bandage folded over with adhesive surfaces in contact. (B) Uncovered adhesive surface.

adhesive surfaces in contact, but leaving $1\frac{1}{2}$ inches of uncovered adhesive surface in the center. The bandage is now about 9 inches long. It is placed on the patient's face, cloth side down, and the center of both eyes marked off. Then two circles the size of a half-dollar are cut out for the eyes. The bandage is now replaced with the cloth side up and the adhesive surface pressed to adhere to the dental compound, and above to the forehead. Pieces of tape 10 or 12 inches long are tied to the corners and then brought back, above and below the ears and tied behind the head.

The bandage is retained for 36 to 48 hours and then removed together with the dental compound and hot compresses are applied, or the author's nasal chamber, used for the same purpose.

178 Lippitt Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOTOLOGY.

Regular Meeting, November 11, 1927.

New Histopathological Findings in the Ear in Lues and Their Importance in the General Pathology of the Ear. Dr. Gustav Alexander.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. E. B. DENCH: The Academy has had from time to time honored visitors, both from this country and from abroad, who have distinguished themselves in one way or another; some as clinicians, others as laboratory men, and still others whose chief work has been along lines of microscopical study. It is rather unusual, however, to have before us a man who is not only a great clinician and an expert operator, but who is also an authority on the microscopical anatomy of that part of the body in which he is interested. Such a man is Dr. Gustav Alexander, to whom we have had the pleasure of listening this evening.

He not only knows his otology better than most of us from a clinical point of view, but he knows his otology as a pathologist better than all of us. Prof. Alexander has not only been an authority on clinical otology for a number of years, but his name during all of this time has also been identified with the microscopical anatomy of the organ of hearing. We, all of us, appreciate what an enormous task it is to study the clinical aspects of certain pathological conditions and then check up on the clinical notes with microscopical evidence.

There are very few in this country who have been able to do this and it certainly has been a great pleasure to listen to Dr. Alexander's remarks on the various pathological changes which may occur in the temporal bone in lues, otosclerosis and cretinism. Most of us are inclined to place the blame for a lack of knowledge in minute pathology on the fact that anatomical material is difficult to secure in this country. While, to a certain extent this is true, I wonder how many in the audience here tonight, given the same opportunities which our guest has had, would be willing to give the time necessary to carry out the character of the work which he is doing? It is not so much lack of opportunity as unwillingness on the part of most of us to do the enormous amount of work necessary in order to achieve such ends.

I was very much interested in the statement made that we sometimes find microscopical changes in the temporal bone, without any clinical symptoms, and also that the changes in lues, cretinism and otosclerosis are in some cases identical. As you all know, the Research Committee of the American Otological Society is very much interested in the investigation of otosclerosis and progressive impairment of hearing from any cause whatsoever, and the fact that changes occur in the temporal bone as a result of the diseases named will perhaps throw some light on the investigation of otosclerosis.

I was also very much interested in what Dr. Alexander had to say about choked labyrinth. The increase in labyrinthine pressure incident to brain tumors or to lues and meningitis is certainly a most interesting condition. We have probably in the past given too little attention to sacculus endolymphaticus and the symptoms which may come from involvement of this structure. In the case of brain tumor, the pressure from neoplasm may cause interference with circulation between the endolymph and the cerebrospinal fluid. This interference may be the result either of pressure from a growth or from the narrowing of the ductus sacculus endolymphaticus by an inflammatory process. It is in pathological conditions of this particular region that the histology of Dr. Alexander will be of extreme value. By the examination of a large number of histological specimens, we gradually learn of the variations from normal in this region, and will certainly discover many conditions, by correlation with clinical history, which will enable us to explain many symptoms whose origin was formerly obscure.

I cannot close my remarks without again conveying to Dr. Alexander my deep appreciation of his kindness in coming here to address us tonight, and I know that I am voicing the sentiments of all those present.

SECTION ON OTOTOLOGY.

*Regular Meeting, Dec. 9, 1927.*Dr. Richard T. Atkins, *Chairman*. Clarence H. Smith, *Secretary*.**Mastoid Complications; Case Reports, etc.** Dr. H. Clifton Luke.*(To appear in a subsequent issue of THE LARYNGOSCOPE.)*

DISCUSSION.

DR. J. R. PAGE: We more often learn more from the cases that die than from those that get well. Not enough of them are reported. Dr. Luke is to be commended for reporting these deaths. He summed up the cases very well, and he criticizes himself, I think justifiably, for leaving the tip in the first operation.

I think I saw the second case with Dr. Luke after streptococci were demonstrated in the spinal fluid and the child had well developed signs of meningitis, and I thought there was little to be done, but suggested some mercurochrome in the spinal canal. I don't know that I mentioned operation on the labyrinth, for the meningitis was so far advanced that it seemed useless. I agree with Dr. Luke, however, that the labyrinth should have been opened at the first sign of any invasion of the meninges, at the first intimation of it, if possible, drain the infection away from them. Both cases were in my clinic, but I think in this particular case it was assumed—and not definitely stated—that the child's labyrinth was active, so it is not known when it became involved. Its involvement was not discovered until Dr. Luke, who had not operated on the case, saw the patient as an ambulatory case. It shows that we ought to be more careful in testing all cases, especially acute cases, in order to recognize at once an insidious complication.

DR. DENCH: I was wondering whether or not in the first case the pus was found in the jugular itself or whether it was an instance where the pus had entered the posterior lacerated foramen and followed the sinus downward into the sheath of the jugular. I know of a case of this kind where the patient recovered after the third operation. I quite agree with what Dr. Page has to say about the advisability of testing the labyrinth in every case. In my service a lumbar puncture is done in every case so that any involvement of the meninges may be discovered. An increased cell count or 4-plus globulin is an indication that the dura has been exposed by disease. Even in cases of acute otitis I think it is wise to take a labyrinthine reaction. In one case where I failed to do this, an impairment, after operation, was attributed to the operation. I think it very possible that this patient's acoustic labyrinth was far from normal before the operation, as a result of an attack of acute otitis many years before.

I feel very certain that the fact that one of Dr. Luke's patients left the hospital so soon after the skin graft had nothing to do with the fatal outcome.

DR. T. J. HARRIS: I agree very heartily with what Dr. Page said about the courage Dr. Luke has shown in reporting these two fatalities. It is true that we learn much more by such failures than by our successes. There is one point, however, about the clinical side that is suggested by these fatalities—that is, that in neither case was there an autopsy. In New York the otologists are open to rebuke for allowing such cases to slip by without autopsies. Dr. Eagleton, in Newark, will get autopsies in almost 100 per cent of his cases, and we fail here in New York. Dr. Eagleton goes personally after his cases, and has assured me that it is not impossible to get them if we only know how to do it. It is up to the chief of the clinic, in a very considerable degree, to follow up these cases. Too often in the hospitals that I have been connected with, the whole thing has been left to the house surgeon, and often the boy is out of the hospital before the significance and importance of getting an autopsy is appreciated, for the undertaker was more on the job than the house surgeon. If it can be done in Newark, we ought to be able to do it here. If we are going to advance scientifically, it is important that we should have a complete record.

Some Mooted Questions in Sound Conduction and Perception During the Course of Chronic Progressive Deafness. Dr. Francis P. Emerson.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. E. B. DENCH: Unless one has read such a paper beforehand, it is almost impossible to do justice to it in discussion. The essayist has touched upon so many points and has presented so many views that any effort to discuss his effort properly will fail miserably. I would like, however, to emphasize a few points. First, the relative infrequency of total occlusion of the Eustachian tube. A number of years ago scarcely a case consulted me whose Eustachian tube had not previously been bougied by some man. At the present time it is seldom that I see a case that requires the use of the Eustachian bougie. I have never seen a Eustachian tube through which I could not get any air, although sometimes the mucous membrane is swollen and air will not circulate clearly to the middle ear. Appropriate treatment will do much for these cases and it is only occasionally that the bougie is required. I am very glad to learn from Dr. Emerson that in a series of over 900 dissections the occlusion of the Eustachian tube was not seen in a single instance.

I most emphatically disagree with Dr. Emerson on the point that we never have middle ear deafness due to obstruction; for instance, I know of cases of serous otitis where the middle cavity is filled with fluid. Such a case is an instance of impairment of hearing due to mechanical obstruction.

I also do not agree that in certain instances an adhesive process is not responsible for impairment of hearing. We may have an adhesion between the malleus and incus which will prevent the proper movement of the ossicular chain or we may have an adhesion between the ossicles and the opposite wall of the tympanic cavity which will also interfere with the movement of the ossicular chain.

One of the great mistakes which otologists make is in assuming that inflation of the middle ear has for its sole object the relief of tubal obstruction. We do not inflate the ear with the catheter simply for the purpose of aerating the tympanic cavity. We use the tube as an avenue through which various drugs in the form of vapor may be introduced into the middle ear, the idea being to stimulate the mucous membrane of the cavity and by thus increasing the blood supply sweep away the result of slowly progressive inflammation. This is of benefit in many cases. In some cases you will find the malleus and incus bound together and the removal of these two ossicles improves the hearing enormously. So I must disagree with the caption of the author that in all cases of progressive hardness of hearing the trouble is with the auditory nerve. The nerve may be affected in many cases by toxemia, intestinal, or due to drugs, such as tobacco, quinin and some of the coal tar products. Or infection may come from the teeth, tonsils, nasal accessory sinuses, etc., but these toxic conditions are not the great causes of progressive deafness. The removal of the adenoids from the nasopharynx has probably improved or prevented more cases of progressive deafness than any one single measure, but this is not caused by the removal of the source of toxemia but by removing a mechanical obstruction.

In my consulting work, I see many cases where interference with the nasal accessory sinuses to improve the hearing is a little less than criminal. Only today I saw two patients with a moderate deflection of the nasal septum. Both had been told that unless this deformity was corrected they would be deaf. Speaking from the experience of many years, I can safely say that I have never seen a single case in which the hearing has been improved by a sub-mucous operation. In the case of tonsils, diseased tonsils should be removed in every case, but I have seen numerous instances where this was urged for the improvement of the hearing and I have tested the hearing of many of these cases afterwards and have found no improvement. That the removal of tonsils and adenoids in early life is a preventative measure against progressive deafness I have not the least doubt, but after the hearing has become greatly impaired, I fear that the removal of the tonsils, even if diseased, will do little to improve it.

I am glad to hear what Dr. Emerson had to say about otosclerosis, its comparative rarity. The Research Committee of the American Otological Society, as you know, are making an investigation concerning otosclerosis and it is

astonishing to find that the disease is so infrequent, when careful examinations are made. On the other hand, I have had many cases come into my office, who have been told that they had otosclerosis, and that nothing could be done for them. While a few of these did have otosclerosis, the majority were suffering from a chronic hypertrophic inflammation. Professor Neumann, when he was in this country a number of years ago, told me that otosclerosis, even in the large clinics in Vienna was comparatively a rare disease, and Professor Alexander recently told us the same thing in this room. Yet many of us see two and three persons each week who have been told that they have otosclerosis, and that their condition is hopeless. Most of these cases are cases of hypertrophic inflammation and can be improved.

We must all remember that there are cases in which otosclerotic changes take place in the temporal bone in patients already suffering from chronic adhesive inflammation of the middle ear, either of a nonsuppurative or sometimes of a suppurative type. In other words, the two conditions are combined in the same individual; of course some of us are inclined to believe that in certain cases the hypertrophic process in the middle ear actually leads to otosclerosis and does not accompany it. It certainly seems significant that the otosclerotic changes in the bone which impair the hearing are in that portion of the bone close to the middle ear. In these cases where the hearing fluctuates it seems inconceivable that this fluctuation is caused by toxemia of the nerve; the fluctuations, in my opinion, are caused by middle ear changes, and when these disappear as a result of treatment the hearing returns to its previous level.

I heartily agree with Dr. Emerson as to the value of the whispered voice as a test. It is the most accurate test that we have, for it is really the human voice that patients desire to hear, so that they may converse with their fellows. Tuning forks are excellent, of course, but only to test the hearing qualitatively. I want to know how low a note the patient can hear, how high he can hear, and whether the bone conduction is increased, lessened or normal, but as a quantitative test the human voice is altogether the best instrument which we have. I might say that in ascertaining the upper and lower limits we are simply obtaining the threshold of some particular tone. But I think they should be taken in every case and I believe the information obtained is much better than that obtained by the audiometer or any other instrument.

Dr. Emerson has mixed me up a little regarding the terms "end organ" and "nerve trunk". In end organ the auditory nerve is the cochlea and when this part of the perceptive apparatus is involved we have good hearing for some tones and poor hearing for other portions of the musical scale and loss of bone conduction. On the other hand, if the trunk nerve is involved, bone conduction is apt to be much diminished but not lost, and this diminution in bone conduction affects principally the forks of the middle register. Where we have a fixation of the stapes we have a classical reaction; elevation of the lower tone limit, marked increase in bone conduction and normal upper limit unless the cochlea is beginning to be involved, in which case the upper tone limits will be somewhat lowered. With this reaction one can practically make a diagnosis of otosclerosis, in almost any instance. I think, therefore, that we must differ from Dr. Emerson when he regards all of these cases as simple cases of interference with the auditory nerve itself.

DR. E. P. FOWLER: Dr. Emerson has demonstrated that he has the courage of his convictions. He has stood for these ideas throughout the years. That is a very valuable trait—to hold one's convictions. If we don't agree with him it is up to us to show where he is wrong, and to prove it; it is not so easy to do that. We have all been brought up with certain fixed ideas regarding progressive deafness and it is hard to get away from them. Let us then criticize this paper point by point and see if we cannot make some progress.

In the first place, I would like to ask Dr. Emerson if he diagnoses nerve deafness purely on the loss in upper tone limits, or whether he uses the relative loss by air and by bone conduction; that is, the loss increasing or diminishing at several frequencies; because unless he does the latter, we cannot say whether he has a true case of nerve deafness or not. Taking the loss at one or two frequencies may not mean anything by either air or bone conduction. I can show cases where the bone conduction is perfectly normal for many octaves, and then a drop of very great magnitude occurs. There is no question, also, but that obstructive lesions may cause the same loss in hearing at the high

frequencies that some nerve lesions cause. I have shown experiments demonstrating this beyond any doubt. For instance, with a weight attached to the drum, we may get at the high frequencies so many sensation units with the head in a vertical position, and with the head horizontal a different amount of loss in hearing units, showing that the changed tension caused the difference. We know that obstructive lesions may cause loss in the high and also in the low frequencies. In many hundreds of cases I have never seen a low frequency definitely diminished without finding all the low frequencies somewhat lowered. It is a waste of time to measure for many low tones, because if you take one or two you know that all the low frequencies are affected.

The number of seconds loss by tuning forks may mean something to one examiner but not to another, for tuning forks may vary greatly. Ten or even fifteen units loss may not mean much of a loss. One of my tuning forks—128 D. V.—will register, say, a 15 seconds loss, another 128 fork 10 seconds, and another 5 seconds in the same ear. These forks are all calibrated and so I can translate all so that they read the same number of sensation units loss. You can do it also with the audiometer and by either method know exactly how many sensation units are lost. The number of seconds lost or the number of feet lost is very misleading. Say the voice is heard normally at 100 feet. Now, if you walk up to within 40 feet and a patient then hears your voice, it really means that he has lost only 10 sensation units. It is very misleading if we confuse the units and the feet, etc. All scales for measuring units of hearing may be reduced to sensation units by calibration.

If we lose 6 seconds by bone conduction, does that mean deafness? No! Not necessarily. The 6 seconds may mean anywhere from 10 to 50 S. U. loss, depending on the forks used.

There are few real stenoses of the tube. Dr. Dench's experience coincides with mine. I think the Eustachian catheter and bougie are very poor instruments with which to diagnose stenosis. The action of the drum is a more accurate index, and this can be determined by manipulation and by swallowing under varying pressures. There are very few tubes that exclude all air from the middle ear.

How many retracted drums return to normal? Don't they all stay retracted more or less, and increasingly so with many exacerbations of the middle ear trouble? The drum is like a derby hat—after it has been blocked into a retracted state a number of times, it will stay retracted; but, as Dr. Dench says, we can change the circulation in the middle ear and can to some extent mobilize the mechanism.

There are three factors affecting conduction through the middle ear—weighting, friction and tension. It depends on whether we get one or more of these, as to whether we get a loss of the high, middle or low notes—or all; and it is very difficult to tell how much you have of each or how much of each one predominates, but you may in many instances be reasonably sure. From tubal closure alone we have never found more than 15 or 20 units loss; that is a small loss. We usually get a good deal more loss in cases of deafness. The doctor and the patient may believe there has seemed no change in the hearing acuity, but one may not always have examined very carefully, and unless we do it very carefully we cannot say there is no loss after an acute exacerbation. After each exacerbation it is commonly found that there has been progression in the deafness. Patients will come down with another infection in the nose, and down will go the hearing. That does not necessarily mean nerve deafness, but the nose is not so far away from the ear, and if you have an inflammation of one of the sinuses you are apt to have inflammation of all the sinuses—and the ear may be regarded as one of the sinuses of the nose. A smouldering inflammation may persist all the time and not be noticed, and until deafness is marked patients don't think so much about the ears.

A characteristic case of nerve deafness: How can we diagnose this differentially from a characteristic case of progressive deafness without nerve involvement? It is often very difficult. I have set up many standards to determine this and almost every one has fallen down. There seems to be no characteristic audiograph curve that seems to give us a lead as to whether or not the deafness will progress. In the thousands of children we have examined in the public schools in the last few years we have not been able to diagnose a single case of otosclerosis or to determine from the ears which are going to

be progressive and which not. We don't know how to tell. I don't know how to tell the difference between a true otosclerosis and an excessive case of immobilization of the middle ear structures—for, after all, otosclerosis is immobilization of the middle ear structures.

The intermittent cases of progressive deafness: I believe they may be due to either nerve or obstructive causes, or both.

Bone conduction: Bone conduction does not aid us much in monaural deafness. Obstructive deafness may be inherited as well as nerve deafness. When there is equal bilateral deafness it does seem to suggest that there must be a nerve lesion somewhere, but we know that obstructive cases also cause bilateral equal deafness, even in the very deaf.

I think it is a mistake to say that the lower tone limit is lowered progressively and comes back in the reverse order. It depends on the forks whether it does or not. I have seen forks where 64 was lost before the 32. It depends on how the fork is made and on the characteristics of the fork, but if the fork findings are translated into sensation units there can be no doubt as to the relative losses. If the tone limits are down a good deal, when they come back they have a further distance to come back than if they were only down a short distance. Therefore, if the lower tones are progressively down, the comeback of the lowest tones seems further, but that does not mean that they come back in reverse order. To avoid confusion, we should make our air and bone conduction measurements in definite logical units of measure (sensation units), and then we can get a true picture as to the sequence in which these changes occur.

When one states that the lower tones are raised, we seem to infer that only the lower tones are less well heard and that the tones above in the scale are heard all right. This is not true. If the lower tones are heard less plainly, some of the tones above will also be (raised) heard less plainly. Audiometer charts show that practically all the tones below 1,000-2,000 are raised if any are (raised) heard less plainly.

After exacerbations in the middle ear, with or without pharyngeal inflammation, there will usually be an increase in the deafness, but there may occur no change in the bone conduction. How can we say that this means nerve deafness? This may occur again and again. The impairment in the health of the nasotubal tissue goes hand in hand with the impairment in the middle ear. I think progressive deafness may be explained in that way—not that these lesions may not at times also cause nerve deafness—nerve deafness is the easiest and most attractive explanation; but we should avoid diagnosing nerve deafness as much as we can in order to better avoid many fallacies.

Dr. Emerson said very truly that evidence points to infection as the starting point in these troubles, and other infections make them worse. I think that is true, but it does not necessarily mean that therefore they all come from nerve deafness. Mechanical changes may and do also cause similar changes in the audiogram. As you know, very loud tones will cause pain, aside from any lesions of the acoustic nerve, so why claim an inner ear or nerve lesion just because certain sounds cause pain? Did anyone ever hear of bone conduction diminishing on account of hypertrophied tonsils, and recovering after the tonsils were taken out? I have never seen it. I don't think we can explain the tonsil cases with deafness in that way, though I think that deafness in some cases may be relieved by tonsil extraction. Even sudden deafness need not be nerve deafness. If not total, it may be due to mechanical causes.

Dr. Emerson spoke about atrophy from nonuse. I think, as he does, that such a notion is false. We have been taught it for years, but absolutely it is not true. He pointed out that it is difficult to have nonuse in the case of the ear, as the sound will reach us through the floor, or cranial bones, or in various other ways.

The calcium content of the blood: No examinations have as yet shown any dependable statistics that indicate the slightest relationship between blood calcium and progressive deafness or otosclerosis; and suppose they do, what does it mean? As far as the beginning of the ear trouble goes, nothing. It started long before the examinations were made. I have made some examinations in children to try to determine its relationship, but without definite success. I think it is best to avoid the diagnosis of nerve deafness unless we use more than the usual criteria—unless we have bone conduction definitely diminished at more than one frequency, and also have a history that will coincide with the

diagnosis. It is better to diagnose the trouble as being in either the middle ear, or in the labyrinth, or in the cochlea, etc. Sound has to travel through these places (or even in the ductus lymphaticus) or in the circulation of the middle ear, or in the lymphatics. But trouble in all of these may occur with or without nerve deafness, at least theoretically.

MR. R. L. WEGEL (Bell Telephone Laboratories): Dr. Emerson's paper is so largely concerned with the pathological side of his subject that I find it rather difficult to do more than make a few remarks on what I take to be some rather minor points concerning the physical aspects. He mentions the whisper test as being the only practical functional test. I presume that as a result of his many years of experience he has found that functional tests are rather minor in importance in making a diagnosis. If he had no other way of making a diagnosis except by means of functional tests, I feel quite sure that he would learn comparatively little from the simple whisper test. On the other hand, if as a result of the rest of his examinations he finds a rather definite indication of the certain kind of impairment, then it is easy for me to conceive how the simply made whisper test will satisfactorily fill in his picture of a particular case. The variety of functional tests which it is possible to make with measuring devices of the present day is very large and undoubtedly each test would add its increment of information for the diagnosis. Tests vary among themselves, both in the amount of information that they can give and in the length of time it takes to make the measurement, so that the number and choice of measurements should vary in any case, depending on the amount of time available for making the diagnosis and the importance of the case. For routine clinical tests Dr. Emerson suggests that the whisper is sufficient. For research purposes, however, this cannot possibly be true.

The whisper is in reality a noise, in that it consists of tones of a very large number of frequencies or pitches distributed more or less at random through the range, perhaps of 1,000 to 5,000 or 6,000 double vibrations per second. A sound similar to the whisper might be obtained by exciting a very large number of tuning forks of different pitches in this range. If the patient were required only to hear a whisper he might be hearing only one or a few of these forks and since the nature of his impairment must be correlated with his acuity for all the pitches this test of course would tell little. The whisper test of course tells nothing of the lower pitch limit nor of the higher pitch limit.

Dr. Emerson also mentions that in bilateral cases there is a tendency for the ears to lose equally in acuity. It is a rather strange thing that even in cases of unilateral deafness the acuity curves are so frequently similar as to be significant. Acuity curves for normal ears of course must always run in pairs; that is to say, the similarities of the curves for the normal ears of one subject are such as to mark them as distinct from curves on other subjects. This would be explained on the basis of the mechanical or anatomical similarity in a pair of ears, but does not seem to explain the similarities of two curves in unilateral cases. It seems that this might have its explanation in the mechanism of the nerve paths from the ears to the brain center. The cochlear branches of the eighth nerve divide before reaching the center and send branches through various ganglia to the other side. If it is possible that nerve fibers at the junction points in the crosspath may, due to lack of complete insulation or in some way, set up impulses in adjacent fibers coming from the opposite ear, then it might be possible that a certain amount of compensation in mechanical sensitiveness of the ears takes place in the nerves themselves. This sort of a supposition, while it seems a bit far fetched, might possibly be used to explain the effects observed.

DR. M. J. GOTTLIEB: I am very much interested in Dr. Emerson's paper and was especially interested in hearing him say that the examination of the hearing in the cases he described gave varying results; that is, that they did not conform to any particular formula. Dr. Drury, of Boston, stated, I think in the *Annals of Otology*, 1926, in an article on otosclerosis, that he believed the prolonged bone conduction in otosclerosis was not always present, and that we should revamp our ideas regarding some of the findings in cases of progressive deafness. It interested me to hear Dr. Emerson say that 100 cases of deafness had the calcium content of the blood examined; also 100 cases of normal people had their calcium content examined; and the result in both instances was 5.5 per cent. It seems to me that we more or less differ as

regards our definitions of progressive deafness, and therefore I doubt whether the 100 cases that Dr. Emerson mentioned were actual cases that involved the labyrinthine capsule.

DR. GUTTMANN: I was very glad to hear Dr. Emerson emphasize the fact that the changes in the function of the glands in the Eustachian tube are responsible for many conditions of deafness. His denial of deafness caused by the conductive apparatus and assumption that every chronic progressive deafness is a nerve deafness is not understood so easily, if we do not agree upon the term—what is understood by the name chronic progressive nerve deafness. We diagnose perceptive or nerve deafness in the main by the functional tests—by a diminished perception for high tones, and diminished bone conduction. Tinnitus, which Dr. Emerson considers as pathognomonic of a nerve disease, may point toward an irritation of the cochlear nerve, but not a disease of the cochlear nerve, as the removal of cerumen from the ear canal may remove tinnitus.

Under the term progressive nerve deafness, Politzer, Alexander and others understand a disease which forms an entity by itself, is found mostly in older persons, shows characteristic functional symptoms, and in postmortem examinations is indicated by characteristic degenerations of the labyrinth and cochlear nerve.

DR. EMERSON: I will take up only two or three points. It is a broad subject and any one of several points might occupy our attention for the evening, but the paper was an attempt to cover the subject in a logical way and show how the deafness started and how it ended, with the hope of stimulating some of the younger men who have, as Dr. Fowler has, facilities for studying tone production and intensities.

As to Dr. Dench's remarks in relation to serous otitis, I agree with everything he said. This paper touched only upon the chronic condition. The acute tubotympanic catarrh is not under discussion. A serous otitis is not under discussion. Any obstruction in the middle ear mechanically obstructs the low notes but after the acute condition has subsided and the low notes have become normal, the further progress of the deafness does not have any relation to the pathology in the middle ear. The etiological factor was emphasized to point out the fact that in every case of deafness, if you examine the glands of the neck during the acute exacerbation, whether they complain of throat symptoms or not, during the first 24 hours, you will find the tonsillar gland on that side sensitive and usually a hyperplasia present. If the acute phase is mild there will be no sensitiveness, but you will note the hyperplasia on the side of the deaf ear. As Dr. Fowler says, the ear is simply an offshoot of the nasopharynx, and any treatment of the nasopharynx affects the blood and lymph streams and indirectly affects the middle ear; so that, in treating the nasopharynx you cannot say that you have not treated the middle ear, even if you did not inflate. However, the hearing is improved by the removal of chronically infected tonsils without any treatment.

Referring to what Dr. Dench said about deviations and operations on the septum, sometimes in extreme deviations the breathing is much better than it seems, and unless you did a submucous operation with the idea of stopping some pathology or improving drainage to relieve infection, the deviation does not count for very much.

Dr. Fowler asked in regard to hearing tests and determining nerve deafness. Of course, the bone conduction test is made very carefully with several forks to determine that point.

Another thing that I wanted to emphasize was that after the acute condition in the tube and tympanum had quieted down the deafness went on without any obstruction in the middle ear and the lowered bone conduction appears at the same stage in the loss of tone perception, whether the process started as a catarrhal middle ear, or as a result of a suppurative process, or in so-called nerve deafness, where there has been no pathology in the middle ear at any time.

In regard to Dr. Fowler's explanation of the return of the low notes to normal, my feeling was that the low notes were raised because the threshold of hearing had been lowered to include the lower part of the scale and not because there was any more obstruction in the tube or middle ear. What he says in regard to the whole tone scale being affected is true, whether in improvement or in loss of tone perception, so that hearing tests are merely relative. What Dr. Miller asks about tension I know nothing about.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Meeting of Dec. 6, 1927.

COLLEGE OF PHYSICIANS.

Cicatricial Atresia of the Larynx. Demonstration of a New Dilating Plug Used in Treatment. Case Presentation. Dr. Gabriel Tucker.

Patient, a girl, age 10 years, while coasting in the prone position slipped forward, striking the larynx on the front crossbar of the sled. Severe injury to the larynx resulted and intubation was done for the relief of the dyspnea. At the end of a week extubation was attempted without success, and tracheotomy was done.

The child was admitted to the bronchoscopic clinic of Dr. Chevalier Jackson, University Hospital, six weeks after the injury, wearing the tracheotomy tube. The larynx showed complete atresia and local evidence of active perichondritis. No effort was made to get through the larynx until the perichondritis had subsided. Efforts to get an opening through the larynx from above were not successful. We were able, however, to pass the smallest size filiform bougie through by retrograde laryngoscopy. A string was passed through the tracheotomy fistula, through the larynx, nasopharynx, and out through the nose; the ends were attached, making the string continuous. The continuous string was used for pulling bougies upward from below in increasing sizes until the lumen was dilated to 18 Fr.

A special plug was then devised from Penrose drain that was pulled into the larynx through the mouth by attaching it to the continuous string. The plug was made larger gradually until at the present time it is the size of the normal laryngeal lumen.

The plug was constructed by folding a piece of the Penrose drain on itself and inverting the unfolded portion of the tube over the folded portion, the strings being attached to either end. When pulled into the larynx it is self-retaining, but is attached to the tracheotomy tube below, and a string is attached to the upper end and passed through the nasopharynx to prevent the plug being swallowed on removal from the larynx.

The pure rubber of the Penrose drain is nonirritating and produces the minimum of reaction to the tissues of the larynx.

The patient had the type of lesion that usually requires laryngostomy, and is obtaining an excellent cure by this apparatus. The patient was shown with the dilating plug *in situ* by Dr. J. Sanford Moyer.

Cerebral Hernia Complicating Chronic Mastoiditis, Perisinus Abscess Thrombosis of the Lateral Sinus and Jugular Vein. Operation, with Recovery. Dr. Henry S. Wieder.

The history of the case is as follows: T. S., male, age 25 years, suffered from chronic otorrhea despite a mastoid operation in Italy in 1914. A week before he came under observation a subperiosteal abscess had been opened. Prior to his operation by the reporter he showed no symptoms or laboratory evidence of serious condition until the day of the operation, when he developed two chills and his temperature rose to 105.8°. There was a leucocytosis of 13,000, but 90 per cent polys. Blood culture before operation showed short chain streptococci. Operation revealed necrotic mastoid and dura, perisinus abscess and pus in the lateral sinus. The jugular vein was tied and severed. Radical mastoid performed and the infected thrombus cleaned out. Patient did well for four days, following which there was a rise in temperature, which was relieved by the release of some pus deep in the wound.

Four days later the neck wound had to be reopened, liberating an ounce of foul pus. Meanwhile optic neuritis had developed in both eyes and there was contralateral facial palsy of the peripheral type. The mastoid wound became filled with a soft, compressible mass, which attained the size of a walnut and filled in almost the entire external canal. While in the process of removing a portion of this mass, which had been hardened by formalin, there developed a cerebrospinal leak, which was finally stopped by packing against the bone at the edges of the wound.

Four days later the skin edges were undermined and brought over the hernia. Healing eventually took place, choked discs and contralateral palsy subsided, the man making a complete recovery. Dr. Wieder then reviewed the various points of especial interest in the case, giving explanations for them, and finally took up the consideration of the various methods reported in the literature of treating cerebral hernia, coming to the conclusion that the method of King was probably the best.

DISCUSSION.

DR. TEMPLE FAY: In my opinion, this case is probably the most interesting one I have read about or had the privilege to see. Cerebral hernia is a condition which we deal with comparatively frequently. The remarkable thing about this case is that Dr. Wieder was able to maintain this patient in such excellent condition in spite of the cerebrospinal leak, and the fact that he washed through from the neck to the mastoid wound after part of the jugular vein was opened. Unless one supposed that the tissues in the neck were opened below the mastoid, then the solution must have passed intracranially. If intracranially by way of the jugular, it is fair to suppose that infection had occurred within the posterior fossa in the sigmoid sinus, and might be responsible for arachnitis at the base of the pons in the cerebellopontile angle.

When Dr. Wieder first brought the man to me I could not determine whether the hernia was of cerebellar or cerebral origin. The treatment of the hernia consisted in the use of a solution of 40 per cent formaldehyde, which caused a hardening and crusting of the surface, and then removing the crust. Spinal punctures in these cases are very dangerous in the presence of infection, due to the possibility of carrying the organism into the subarachnoid spaces. Dehydration is much more effective. The water intake must be cut down considerably, an intake of not more than 300 to 500 c.c. in 24 hours. With the addition of magnesium sulphate by mouth we were able to reduce some of the hernia and procure a dry wound. This was important during the time of healing of the secondary closure. The man at the present time has no focal brain symptoms, though there is still some evidence of intracranial pressure. It is my opinion that nothing further should be done for the present, as he is practically symptom free.

DR. GEORGE W. MACKENZIE: I want to congratulate Dr. Wieder on his very full report and the excellent results he obtained. These cases do not occur very often, and when they do they tax us considerably. It has been pointed out by MacEwen, Koerner, Politzer and a number of other investigators that intracranial complications rarely occur singly. Dr. Wieder, did you determine the cause of the greenish colored pus?

The seventh nerve involvement of the side opposite the ear involvement is a rather odd complication. Someone reported a similar case before this Society about a year ago. Do you remember whether all the branches of the seventh nerve were involved? Were the muscles in the upper part of the face involved as well as the lower?

With reference to the cerebrospinal fluid that seemed to transude through the brain substance, ordinarily in the case of hernia the pressure around the edges of the opening in the bone is sufficient to prevent the escape of the fluid from the subarachnoid space surrounding the hernia. Was the Tobey test for thrombosis tried out before the operation?

DR. PHILIP S. STOUT: I was pleased to hear that Dr. Wieder lost little time in tying off his internal jugular vein in the neck as soon as he found the abscess around the sigmoid sinus and pus in the sinus itself. Very prompt ligation of the internal jugular vein low down in the neck undoubtedly is a life saver and the removal of part of the vein between two tied ends is an additional advantage. Dr. Wieder and his associates are to be congratulated on the outcome of this very unusual case.

DR. BENJAMIN SHUSTER: One thing about this patient that should be considered and the patient warned is the possibility that he might go to another doctor who would mistake the swelling for an abscess and thrust a knife into the tumor back of the ear, not knowing the previous history. Another point, the fact that this tumor or hernia formation still exists might be indicative of pressure intracranially and a latent abscess, for instance, might still be present.

DR. WIEDER (closing discussion): Prager's iodine was used. One 20 c.c. ampule was given intravenously every day. With reference to the contralateral

facial paralysis, I did not feel that it could be due to a coincidental facial palsy since the man was in bed and he was sheltered from all cold, etc., but the neurologists insisted it was peripheral, so we bowed to their judgment. We did not do the Tobey test, as we felt we could make a diagnosis of lateral thrombosis without it. This test is not without its dangers. I saw one traumatic case in which there was some question as to infection develop meningitis just as soon as the spinal puncture was done.

An Unusual Type of Diplacusis Dr. Douglas Macfarlan.

The case herewith cited is one which occurred to the author and presented an unusual opportunity for study.

During an acute attack of purulent rhinitis of the ordinary "head cold" variety, there developed bilateral Eustachian catarrh, stuffiness in the ears, impaired hearing, a "sea-shell" tinnitus, transitory spell of dizziness and diplacusis. The dullness of hearing, diplacusis and tinnitus were constant for two periods, lasting three to four days each; there was a remission of symptoms for a week between these two attacks. There has been no previous ear trouble.

As all the ear symptoms appeared simultaneously, the diplacusis was immediately manifested. The false note was heard only in the left ear and took the pitch of the second semitone above the true tone. It was the left ear which was somewhat more deafened than the right ear. The hearing tests showed findings of the acute incipient catarrhal type—bone conduction lengthened above normal in each ear, air conduction proportionately decreased. Weber referred to the worst ear, and a high plus-minus Rinne, left; high plus, right.

B488 Koenig fork was interpreted as D581 in the left by reason of false impression. The fork was struck by a standard blow, a swing pleximeter, and was timed by a stop watch from the time struck. Normal for this fork is 30 seconds B. C., 55 seconds A. C.; dampening factor expressed in sensation units per second is 2.0, the K for reducing hearing loss to percentage is 0.81.

The peculiar feature of the case is the type of diplacusis instead of being a quarter or a semitone above the true, as in the majority of cases cited by others, it was two semitones above. Thus listening to single tones, the perfect harmonis compliment was heard in the left ear. The effect was one of a duet, music whistled or sung by the subject gave the same effect as was noticed in listening to others. The range of this false impression existed only between the pitches F345 and E647; the hearing above and below these pitches was true.

The false note in all instances was complimentary, taking the ratio of 1:4 in the logarithmic scale. "The logarithmic scale of frequencies is one in which the spaces are proportional to the ratio of the frequencies; it is represented in actual sounds by the successive tones of the pianoforte. For instance, the successive octave points represent tones having frequencies in the ratio 1:2:3:8:16, etc.; the ratio is the same throughout, and the actual distance in inches from any note to its octave on the scale, and also on the piano keyboard, is the same, whether the note is on the lower part of the scale where the number of vibrations is small, or whether it is on the upper part of the scale having several thousand vibrations per second—hence the distance between any two specified partial tones is constant throughout the logarithmic scale."

All symptoms cleared up spontaneously within two weeks' time, coincident with the disappearance of the "head cold".

DISCUSSION.

SHAMBAUGH and KNUDSEN report ten cases of diplacusis, of which seven showed the false hearing above, three below the correct tone. In six cases the effect extended over the entire tonal range, from 60 d. v. to 4,800 d. v. The most conspicuous finding is that in all of the cases there was a positive diagnosis of internal ear disease. In not a single case was there any evidence of a middle ear disease that could be held responsible for this symptom. The conclusion is, that diplacusis is characteristic of internal ear disease.

It appears that the case here cited had some congestion of the inner ear as responsible for the diplacusis and dizziness. The picture, however, was one of an acute aural catarrh of a transitory nature. There were inner ear as well as middle ear symptoms. For the most part, Shambaugh's cases showed pitch differences of quarter and semitones; there is no mention in his reports

or in others investigated of such exact harmonic relations. (If overtones have frequencies which are exact multiples of that of the fundamental they are called harmonics, otherwise they may be designated as inharmonic partials—Miller.) In our case the false note was invariably two semitones above.

Shambaugh's reasonings in his cases are so logical as to merit repetition. "To the physiologist the problem of particular interest has to do with the type of pathological changes in the cochlea that are responsible for this interesting symptom, and the conclusions that may be drawn from its occurrence regarding the theory of sound perception. One thing is apparent, the ordinary degenerative changes from whatever source do not cause this symptom since it is only the exceptional case of internal ear disease where it occurs." Assuming the rationality of his theory that the membrane tectoria is the responsive mechanism in sound transformation, Shambaugh states: "It would seem from its sudden onset that the symptom of diplacusis is probably due to some pathological deposit adhering to the tectorial membrane; for example, a blood clot or a fibrinous deposit; such a deposit would influence its vibrating qualities so that a segment that normally vibrates to a certain tone will now vibrate to a tone just below or just above this tone.

It would seem that the transitory character of the diplacusis in our case might readily be explained upon such lines, yet the exactness of the harmonic relations exhibited over nearly an octave would require an exactness of deposit of a fibrinous deposit such as is improbable. The demarcation limits of our diplacusis were rather sharp, and just beyond these limits the tones were again true. The exact pathology is still conjectural.

There are two problems now occupying the attention of educators, which problems should be of concern to all otologists: the hard of hearing child and the deafened child.

1. The Hard of Hearing Child: The American Federation of Organizations for the Hard of Hearing has sponsored an investigation into the number of school children handicapped by deafness. On the basis of a 6-to-9 sensation amount in loss being a handicap, there are 14.4 per cent of the school children who are hard of hearing. In terms of distance for hearing speech, this means that those who cannot hear the speaker until they come nearer than two-fifths of the average hearing distance are considered to have abnormal hearing. Fourteen per cent! The figures are much larger than were suspected. In one locality, out of 4,112 pupils tested, 595 would be classified as deficient in hearing; 3.2 per cent having defects in both ears, and 11.2 per cent in one ear only. Contrary to the popular belief, the child having one defective ear was found to be handicapped, and likely to be retarded. (School Health Studies, No. 13, July, 1927, Bureau of Education, Department of Interior, "The Hard of Hearing Child.")

Estimating the public school children of the United States at 24,000,000, it would indicate that over 3,000,000 had some hearing defect.

The cost of the school child repeating the school (as most of these children do) makes the expense of this handicap run into millions annually.

The practical consideration, or the problem of "what to do about it", suggests the early control of the ear complications of infectious fevers, the removal of obstruction to Eustachian tube aeration, the prompt attention to tonsil and adenoid hypertrophy, and to nasal catarrhs.

2. The Deaf Child: About one-half of the 17,000 children in the schools for the deaf have early acquired deafness. A considerable number of cases could be saved from this permanent and costly handicap. The annual expense of educating a deaf child runs into the neighborhood of \$1,200. The education period is about twice as long as with the normal child, and the end results are scarcely ever as satisfactory. Some years ago there were \$27,000,000 tied up in the deaf school "plants". The annual budgets ran around \$18,000,000.

The National Research Council is now completing a survey which should be very valuable. Many questions will be brought out, one of which is the possibility of utilizing for education the residual hearing that most of the children have.

DR. J. A. BABBITT would like to ask Dr. Macfarlan his method of procedure in determining whether a child 1 year old was deaf or not?

DR. DOUGLAS MACFARLAN: Dr. Babbitt has put a very difficult question. I should answer by, "Ask me another".

The determination of deafness depends so much upon subject. We response rather than upon objective examination, that it is obvious we can discover the probable condition of hearing in the infant. We must see if they response to noises; they can tell us nothing. An observant mother will give the best reports. Ask if the child notices the door bell, or if the mother has seen the child jump when an automobile muffler has exploded, or has the child's attention been changed when an automobile horn has been sounded, and the like. Remember that many loud sounds actually produce tactile sensations, and attention to these tactile stimuli may be mistaken by us for hearing.

To illustrate how a deaf child is examined, I will cite the case of a doctor's child who was brought, diagnosed deaf by several capable otologists. The child was very intractable, shy, suspicious and fretful. A phonographic audiometer that I have constructed was set at 30 per cent hearing loss. The receiver was placed on the table, and the mother was left alone in the room with the child. A pantomime of listening to the telephone was copied by the child, and it was soon discovered that the child recognized the moment the machine was stopped. At 30 per cent loss the child should hear language, so we knew that speech instruction by the aural method could be started at once. With an efficient teacher the child has, in two weeks, learned a considerable number of words and sentences. The "deafness" in this case turned out to be inattention.

Dr. Mackenzie has referred to the palpebral reflex as a sign of hearing response. This sign I have been particularly studying in the testings made for the National Research Council Survey. I find it most independent. It is very often absent when it should be present; again, it is present as a response to heavy tactile stimulus of air-borne sound. This sign bears the name of Dr. Gault in Hay's textbook; this and other ocular signs have been well written up by Otto J. Stein. (Three Reflex Signs Useful for Examining the Ears for Deafness; Transactions read O. and O. L., 1919.)

DR. JAMES A. BABBITT: A suggested method was reported to me by friends interested in this in Chicago, and applied in a foundling institution. A coincident sharp rap on the feet and concealed noise by the ear was repeated a number of times. Later the rap on the feet was omitted, but the customary cringing of the child, who expected the blow from the noise that was made, convinced that hearing was present.

Otorhinologic Hemorrhage and Some Methods of Control Dr. Philip Stout.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. WM. H. DEARDORFF: The question I wish to ask is, "What is done when the entire surface of the tonsillar fossa presents a general ooze and you cannot find the bleeding point?"

Dr. Deardorff in answer to his own question stated that he believes in the procedure of placing the sponge between the pillars and suturing to retain the sponge in place but does not feel that it will be sufficient in every case.

DR. SAMUEL SKILLERN: I am sorry that I cannot agree with a number of Dr. Stout's statements in regards to his methods of the removal of tonsils.

In the first place, Dr. Stout advises the use of dull instruments; then he states that he has about 6 per cent severe hemorrhages following his tonsillectomies. In the Skillern Clinic we use the LaForce method for the removal of tonsils, for all cases that can be taken out by this method, and we have about 0.5 per cent hemorrhages. The dull blade of the LaForce crushes and so traumatizes the vessel before it is cut cleanly by the sharp blade that clotting occurs in these vessels very quickly.

In those cases in which it is impossible to engage the LaForce instrument, which is about 5 per cent, we use sharp knives for the dissection. We all know how dull instruments tend to tear instead of cut, and torn tissue will always bleed more than that in which a clean cut has been made. In a clean cut vessel bleeding is checked by the contraction of the vessel, the retraction of the vessel into the soft tissue and by the closing over of the bleeding vessel by the surrounding tissue.

As to checking hemorrhages, the general muscular ooze from the fossae which sometimes occurs is controlled by placing a small sponge directly into the fossae. This sponge will often be held in place by the anterior and pos-

terior pillars folding in over it; if this does not occur it is held by some suitable forcep. We do not make pressure on this sponge as we believe pressure holds the blood out of the cut vessels, and when the pressure is removed bleeding will continue, whereas the sponge lying in the tonsillar fossae acts as a foreign irritant and causes a normal contraction of the cut tissue. It also absorbs the fluid element of the blood, which allows a more rapid coagulation and at the same time keeps the throat free of blood. If this sponge becomes saturated it is removed and a dry one replaced. If there is no *active* bleeding from a vessel the first sponge generally does the work; if not, a second will nearly always stop all ooze.

The actively bleeding vessels, if a vein, is picked up by a hemostat and crushed; the arteries are picked up by a hemostat which is allowed to remain on for several minutes if after its removal the vessel continues to bleed it is ligated, very occasionally sutured.

We never sew the anterior and posterior pillars together, nor do we ever sew a sponge in between the pillars as we do not consider this good surgery.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Semi-Centennial Meeting of the American Laryngological Association will be held in the Hotel Raleigh, Washington, D. C., on May 1, 2 and 3, 1928. The guest of honor will be Sir St. Clair Thomson, of London, England, who is an Honorary Fellow of the Association. A number of papers will be devoted to the achievements of Laryngology during the last half-century, both in civil life and in the army and navy during the World War. All members of the profession interested in Laryngology are cordially invited to be present.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, Inc.

The next Annual Meeting of the American Laryngological, Rhinological and Otolological Society will be held at Washington, D. C., on May 3, 4 and 5, 1928, immediately following the meeting of the Congress of American Physicians and Surgeons.

The meeting place and headquarters will be at the "Raleigh".

Rooms will be assigned in the order of the receipt of reservations and should be made as early as possible. It would be appreciated if you would make your reservations direct to the Raleigh.

Due to the early date upon which the meeting is to be held, it is necessary that those desiring to present papers should notify the Secretary immediately in order that the program may be completed and printed in good season.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

The Santa Barbara meeting of the Pacific Coast Oto-Ophthalmological Society will be held April 18, 19 and 20, immediately preceding the meeting of the Western Section of the Triological Society. Plan to be there Wednesday, Thursday and Friday and, if possible, stay Saturday for the meetings of the Triological Society.

The scientific program will run from 9 a. m. to 1 p. m., leaving the entire afternoon for golf, automobile rides, etc.

The following men will be guests of the Society and will take part in our program: Dr. Norvil Harvey Pierce, Chicago, Professor, Laryngology, Rhinology and Otolology, University of Illinois; Dr. John F. Barnhill, Indianapolis, President of the American Laryngological, Rhinological and Otolological Society; Dr. Luther Peter, Philadelphia, President, American Academy of Ophthalmology and Oto-Laryngology; Dr. Henry H. Tyson, New York City.

